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Saint Joseph's Hospital Issue

ST. JOSEPH'S HOSPITAL

HISTORY OF SAINT JOSEPH'S HOSPITAL

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ADMINISTRATOR

The story of St. Joseph's Hospital evolves through 3 distinct phases: there was its humble inception, necessitated, as was so often the inciting factor, by the events of the period, followed by 92 years of unceasing, selfless service; and the third phase must rest in the inevitable future reconstruction which shall provide for continued competent medical care for the City of Baltimore and its surrounding communities.

It was in the Civil War period, in November 1864, that Mrs. Catherine Eberhard, of Baltimore, donated 3 two-story houses, located at Nos. 187, 189, and 191 North Caroline Street to the Reverend Mother Mary Agnes of the Third Order of St. Francis, Philadelphia Foundation, and designated that these structures be used for the care of the sick and infirm. Three Sisters of St. Francis were assigned to administer this charge; these were the Sisters Clara, Scholastica and Crescentia, and the latter served as Superintendent until the year 1888.

The original three houses functioned as a hospital and patients were continuously served until 1867, when demands for increased facilities necessitated the purchase of a tract of land, the present site of St. Joseph's Hospital, from Noah Walker, Esquire. At that time, a temporary building capable of accommodating about 50 patients was erected, and an additional number of Sisters was assigned to administer the growing needs of the hospital. In April 1871, the Sisters of St. Francis in charge of the hospital were made a corporate body of the City and State, and in October of the same year, the cornerstone of the present structure was laid. From 1864 to 1871, nearly 700 patients were cared for in the original 3 houses and the temporary structure which followed.

As has been mentioned, St. Joseph's Hospital was founded at a time when Baltimore was experiencing the consequences of Civil War and the military element was a factor in the further development of the hospital. From 1881 until the opening of the Marine Hospital in 1887, St. Joseph's was the hospital selected by the Government to care for the sick and wounded sailors entering the port of Baltimore. In 1896, a new wing was added, and these increased facilities were greatly taxed by the influx of wounded soldiers of the Spanish-American conflict.

Originally, the medical care of the patients was entrusted to Doctors Doyle and Trautman. In 1870, Dr. Oscar Coskery was appointed Medical Director and served until his death in 1889, being succeeded by Dr. Ferdinand E. Chatard. The original Medical Staff consisted of: Chief of Staff; Dr. Oscar Coskery; Consulting Staff: Doctors N. P. Smith, F. E. Chatard, Sr. and Richard McSherry; Active Staff: Doctors I. C. Chew, F. E. Chatard, Jr., G. L. Robinson and S. E. Atkinson.

The story of the development of St. Joseph's Hospital would not be complete without at least a brief biography of Dr. Francis J. Kirby. "Professor" Kirby, as he was known to four and five generations of many Baltimore families, was born in 1869 and graduated in 1892 from the Baltimore Medical College (which in 1913 merged with the University of Maryland School of Medicine). Upon his graduation, Dr. Kirby was appointed Resident Physician at St. Joseph's Hospital and served on the Active Surgical Staff until semi-retirement in 1951. Dr. Kirby was on the Surgical Staff of most Baltimore hospitals, the Annapolis Emergency Hospital and St. Mary's Hospital at Leonardtown, Md. He was a member of the American College of Surgeons and was later honored by election to the International College of Surgeons.

At the end of the last century, care of patients was performed solely by the Sisters. The added burden of the sick and wounded soldiers would have placed an impossible demand on the Sisters, had it not been for a volunteer group of heroic women, led by Mrs. Ferdinand E. Chatard, who daily devoted many hours to actual bedside nursing. In 1901, these women organized the Woman's Auxiliary of St. Joseph's Hospital.

In response to this need, also in 1901, it was determined to open a training school for nurses. Sister Mary Bertrand was appointed Superintendent of the Nurses' Training School, which graduated in 1904 its first class of five: the Misses Frances M. Branley, Rose M. Carney, Mary E. Cloman, Anna E. Cunningham and Etta S. Rayle. The longest surviving member of that original nursing class was Miss Branley, who died in December 1956, in her native Ireland. Miss Branley's nursing life paralleled the de-

velopment of the era of modern medicine since the early 20th century. Following graduation, she remained at St. Joseph's Hospital and served as Head Nurse of the Operating Rooms until World War II. In 1918, she served with the University of Maryland Unit in France, supervising a Base Hospital Operating Room directly behind the front line. Following the war, she became Assistant Director, later Acting Director, of Nurses at the University of Maryland Hospital. She returned to St. Joseph's Hospital and was Directress of the Nurses' Training School from 1922 to 1945 at which time she returned to Ireland and spent her remaining years in volunteer nursing and Church work.

The modern concept of intern and resident training began in 1912 with the appointment of Dr. Edward S. Johnson as Resident in Surgery. Dr. Johnson's recent death brought sadness to the entire community, and especially to his many associates and friends within Medicine. There is no fitting tribute to 45 years of skilled and courteous service.

Upon organization of the American College of Physicians and Surgeons, St. Joseph's Hospital re-organized its Medical Staff and received the approval of the American College in 1923. A new Nurses Home was built in 1924–1925. In 1927, the operating suite was relocated and enlarged to include its present 5 major operating rooms, a minor surgical room, and a room for broncho-esophago-gastroscopy.

Further re-organization of the Medical Staff was accomplished in 1940. Medicine and its selected specialties was headed by Dr. Francis J. Geraghty; Dr. William R. Geraghty became Chief of Surgery, and Dr. Thomas K. Galvin, Chief of the departments of Obstetrics and Gynecology. This re-organization program was approved by the American College of Physicians and Surgeons for the training of residents in these specialties.

In 1941, the hospital Red Cross Unit was formed under the direction of Miss Florence Laroque. These unselfish ladies meet weekly and contribute materially to the efficient and economical functioning of the hospital. In 1952, under the sponsorship of the Woman's Auxiliary, a Coffee Shop was opened as an added convenience for hospital visitors and personnel.

The third phase of the story of St. Joseph's Hospital may have begun in January 1956, when the very existence of the hospital was threatened. The value of St. Joseph's Hospital to the community may be exemplified by the following few statistics for the year 1955. The total number of admissions was 7554; of these, 1858 patients were service patients and 890 cases were admitted from the Accident Rooms. There were 12,632 patients treated in the Accident and Emergency Rooms and an additional 10,100 Out-patient Clinic Visits. Major and Minor Surgical procedures in the Operating Rooms totalled 3755.

Because of the extreme age of its physical plant, the hospital administrator was advised by the Maryland State Department of Health that many improvements, especially the installation of modern fire prevention equipment, were necessary if the hospital were to continue to function, and that these alterations should be accomplished in anticipation of the construction of a new, modern hospital.

In June 1956, the Medical Staff appointed a committee composed of Dr. Otto C. Brantigan, Chairman; Dr. Harry J. Connolly; and Dr. John J. Krejci. This committee was instructed to work with the administration to take those steps necessary to the continued and effective functioning of the hospital, until the time when a new physical plant could be erected. The first function of this committee was the formation of an authoritative lay board. At the present time, this lay board consists of 21 members who very willingly give their services. These men are representative of every walk of business and industry, and their chief qualification for membership consists of the fact that each, without regard for personal business interests, religion, etc., is willing and anxious to work for the advancement of St. Joseph's

Hospital. The members of this board of St. Joseph's Hospital are as follows: T. Gordon Bautz, Mrs. Geo. H. Brouillet, M. H. Ellingsworth, Robert C. Embry, J. Early Hardesty, J. Lee Hooper, Morton L. Kemper, W. H. Kirkwood, Jr., Gilford F. Kunz, M. Oldham Lewis, Chairman, J. Irvin McCourt, Wm. E. McGuirk, Joseph B. McLaughlin, John N. Mackall, J. Jefferson Miller, Thos. F. Mullan, Sr., Wm. J. O'Meara, Joseph J. Rochlitz, W. Thomas Swomley, Craig E. Taylor, and C. Howard Timanus.

In addition to the formation of an authoritative Board of St. Joseph's Hospital, many other steps were taken to assure improved service. The Medical Staff was completely re-organized; a sprinkler system and fire doors are being installed and, of course, fire drills continue to be held at regular intervals. Since parking in the yard of the hospital was considered to be a fire hazard, parking has been banned. Throughout the hospital, the changes are evident; new ice machines have been placed on each ward, the kitchen has been remodeled and great expense incurred to provide new equipment. The wards are better lighted for both improved medical care and for the reading comfort of the patient, by conversion from direct to alternating current. Other less important steps have been taken to assure a continued high level of service to the patients of St. Joseph's Hospital.

It is hoped that the summary of this brief history of St. Joseph's Hospital is in reality not a summary, but the expressed hope of a beginning, in that plans are being made and vigorously pursued to insure the purchase of land and the construction of a new modern hospital which for an additional 92 years will serve the people of Baltimore and its environs.

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ARTICLE OF INTEREST

ELECTROSHOCK THERAPY IN A GENERAL HOSPITAL

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As psychiatry comes of age, it is able to take its place more easily among the other medical specialties in healing the sick. In its infancy, like human infants, it had a very limited range of activity, being largely confined to mental institutions. In its adolescence, like human adolescents, it was inclined to be noisy and overenthusiastic about its own importance, often annoying those in the older specialties by its extravagant claims and frequent dearth of visible accomplishment. Now, as a more mature specialty itself, psychiatry is able to take its proper place on the medical team, to provide better total care for the sick in any hospital or community. With better and faster diagnostic and therapeutic methods available than formerly, psychiatrists can now help many patients in a general hospital who formerly would have been treatable only in mental hospitals, thereby eliminating much of the expense and stigma formerly associated with mental illness.

General hospitals are becoming gradually more aware of their opportunities to provide treatment for those sick with mental and emotional as well as physical illness. Formerly, psychiatric patients were not welcome in general hospitals, probably because of the practical problems involved in their care, such as the special facilities and personnel needed, and the length of time necessary for any substantial improvement. The problem of adequately controlling noisy, overactive, or suicidal patients in a general hospital is of course great. Many psychiatric patients still cannot be adequately treated except in a properly equipped mental hospital, often over a period of many months or even years. But with speedier modern methods of psychiatric treatment, a short stay in a general hospital will restore many to mental health who formerly would have had to endure the difficulties, expense, and stigma of a mental hospital.

Depressions are particularly benefitted by modern methods of psychiatric treatment, such as electroshock therapy. As depressions constitute one of the commonest forms of mental illness, and as electroshock therapy greatly shortens the course of the illness, it means a tremendous saving of time and money if facilities are available to treat depressed patients in this way. It means not only a saving of the cost of mental hospital care, but also a saving of the self-respect of the patient and his family, if such treatment can be carried out in a general hospital like any other treatment for any other illness. Depressions are frequent whenever the demands of life exceed a person's capacity for meeting those demands, and are particularly frequent at times such as the involutional or the later years of life, when the demands of life are particularly difficult and the emotional resources limited. To delay treatment for such depressed patients is to risk death by suicide. A week or two in a general hospital, with prompt electroshock therapy, can restore these depressed people from complete incapacitation to health sufficient for meeting the ordinary demands of life.

The most important factors in the success of electroshock treatment in a general hospital have been the proper selection of cases, the competent application of the treatment, and

the promptness with which treatment is started. In mental hospitals, a newly admitted patient is usually observed for a week or more before starting treatment. In a general hospital, because of the greater danger of suicide, prompt evaluation is essential, and treatment should be commenced within the first day or two following admission. Treatment is then continued until the depression has cleared up, together with as much psychotherapy as possible. Arrangements must of course be made for continued observation and psychotherapy after leaving the hospital, to increase the patient's efficiency in dealing with the situation that originally produced the depressive reaction, and to prevent any possible tendency to relapse on returning home.

The following cases, all treated by me at St. Joseph's Hospital, are selected from many others as illustrating better than statistics the results obtained in the electroshock treatment of depressed patients in a general hospital.

A) *Depressions:* As mentioned above, depressions are greatly benefited by electroshock therapy, as shown by the following cases.

Case 1: T.B.B., a 65-year-old clerk, became depressed following the death of his only son two years previously. He complained of a lump in his throat, a constant feeling of tightness in his chest, a tight feeling around his head, and a lack of interest in the activities that formerly interested him. He felt too exhausted to do his work properly, resented his wife's efforts to help him, and saw no point in continuing to live. He showed no response to a trial of office psychotherapy, with vitamin and amphetamine medication, so was admitted to St. Joseph's Hospital, where he was given three electric shock treatments in the course of his one week in the hospital. At the end of that time, he had no more depression, no more symptoms, and was able to return to work. He continued office psychotherapy for several more months, to help him to accept the realities of his life better, to see his assets as well as liabilities, and to fill his life with new interests to make up for what he had lost. He has continued well for three years since then.

Case 2: N.K., a 52-year-old unmarried telephone-operator, complained of inability to sleep for more than a few hours each night, for the past eight months, with feelings of weakness, tiredness, and discouragement, and lack of interest in anything. She had made one suicidal attempt, in spite of her strong religious conviction that suicide was never justified. She was admitted to St. Joseph's Hospital, and was given four electric shock treatments in the course of ten days, with excellent relief of her depression. Following this she was seen occasionally in the office for several months more, to make sure that there was no return of her depression, and to help her to deal more effectively with various personal problems in her life. She has remained well for the three years since her last treatment.

Case 3: M.M., a 59-year-old widow, had been depressed and unable to sleep for the past three years. She had been treated by at least six other physicians, including two other psychiatrists, without benefit. She had had a variety of medications, hormones, and even several operations, but only felt more discouraged when none of these therapeutic efforts helped her. She had always been an energetic perfectionistic person, who had brought up four children after her husband's early death, and she was not happy in the life of inactivity that followed her children's growing up. She felt useless and lonely in her big empty house, but feared that she was too old to start making a new life for herself. The only romantic interest in her life had faded as she had become more depressed, as her admirer was no longer attracted by the melancholy person she had become. After a year of office psychotherapy with no appreciable improvement, she was admitted to St. Joseph's Hospital, where she was given 14 electric shock treatments in the course of a month, with dramatic benefit. Her depression disappeared, she was able to sleep

again without medication, her enthusiastic interest in life returned, and she was able to readjust her life to give her more satisfaction and feeling of accomplishment. She has remained well, cheerful, and able to lead a normal active life for the four years since her last treatment.

Case 4: E.L., a 63-year-old Swedish carpenter, with a history of several previous episodes of depression, had become depressed and agitated during the last two months, unable to sleep, eat, or to take any interest in his home or work. He spent his time brooding about the sins of his youth and blaming himself bitterly for mistakes he had made 40 years ago. He was unable to relax, and would pace the floor, wringing his hands and moaning to himself, not responding to the efforts of his devoted family or his family physician to help him. He was admitted to the hospital, electric shock treatment was started the day of admission, and he was able to go home in a calm, relaxed, and happy frame of mind one week later, after four electric shock treatments. He has remained well during the year that has now elapsed since his last treatment.

Case 5: W.S., a 70-year-old clerk, was admitted to the hospital following a fall in which he fractured his left hip. An orthopedic operation was done, to immobilize the fractured fragments, and during the week following the operation he became gradually more depressed. When seen in consultation two weeks after operation, he was very depressed, sitting silent, unable to take any interest in anything going on about him, unable to eat or sleep well, and expressing ideas of hopelessness about ever being able to do anything again because of his injury. Six electric shock treatments were given, during the next two weeks, with complete relief of his depression. He became cheerful, talkative, able to take an interest in his surroundings, and able to talk about the future with confidence. In the year following his last treatment, he has remained well and happy, without any further depression.

B) *Psychosomatic Cases:* Some cases that are admitted to the hospital because of psychosomatic accompaniments of depression respond quickly and satisfactorily to electric shock treatment, with subsequent psychotherapy, as shown by the following cases:

Case 6: H.H., a 56-year-old widow, was admitted to the hospital because of persistent vomiting for the past month. This vomiting had persisted in spite of the treatment given her at home by her family physician, and by the time of her admission to the hospital she was exhausted, emaciated, and too weak to get out of bed. All studies were negative for organic disease, except for some hypochromic anemia secondary to her starvation. When seen in psychiatric consultation, it was found that her symptoms had developed in a setting of overwork and emotional strain caused by the necessity of caring for a daughter crippled by severe arthritis for the past four years. She had become more tired and more depressed as she tried to cope with this unfortunate home situation without help, but had forced herself to keep on until prostrated by the psychosomatic vomiting which represented her inability to "stomach" an intolerable situation. A week of bed-rest, medicinal treatment, and psychotherapy failed to produce any appreciable improvement, so she was started on electric shock treatment, with dramatic benefit. After five treatments, she was cheerful, calm, able to eat well without nausea or vomiting, able to sleep well, and able to make constructive plans for meeting the difficult home situation in a more realistic manner. There has been no recurrence of her depression or her vomiting in the three years since her last treatment.

Case 7: M.A., a 65-year-old housewife, was admitted to the hospital because of difficulty in swallowing for the past few months. Because of this difficulty, she had reduced her diet to liquids, had lost much weight, and felt weak, exhausted, and unable to do any of her usual activities. Examination showed hypertension,

electrocardiographic evidence of myocardial degeneration secondary to coronary arteriosclerosis, and some secondary hypochromic anemia. She failed to improve on bed-rest and medicinal treatment, so her family physician requested electric shock treatment to try to relieve her depression and its accompanying psychosomatic symptoms. Eight treatments were given, with excellent results. She became cheerful, regained her appetite, and no longer showed any evidence of being unable to swallow. She regained weight and strength, was able to resume her usual housework, and has been free from psychosomatic symptoms for the two years since the last treatment.

Case 8: T.V., a 49-year-old Italian-born widow, was admitted to the Surgical Service of the hospital because of persistent indigestion which was thought to be due to gall-bladder disease. X-ray and other studies showed no evidence of organic disease, but she continued to complain of abdominal discomfort. On psychiatric consultation, it was found that her symptoms had developed in a setting of over-work and emotional strain at home, where her life was complicated by a senile tyrannical mother and several children who caused her much worry by not living their lives the way she thought they should. There was much loneliness, financial worry, and lack of recreational activity. The result was an agitated depression, with psychosomatic symptoms that expressed the pain caused her by the various intolerable situations in her life. Five electric shock treatments in the course of two weeks in the hospital, produced a dramatic improvement, so that she became cheerful, gay, active, and free from symptoms of any sort. She was able to return home, meet her problems more realistically, and has been well for the five years since her last treatment.

C) *Schizophrenic Cases:* In general, schizophrenic patients can be better controlled in a mental than a general hospital, but an occasional case can be adequately treated in a general hospital.

Case 9: A.J., a 66-year-old housewife, was brought to the hospital because of inability to eat, talk, or walk. For five years, she had lain silent in bed, having to be fed and cared for as if she were a baby. Neurological examination showed no evidence of organic disease of the nervous system, and no abnormalities except for some disuse atrophy of the legs. Mentally, she was very withdrawn, lying silent, with her eyes closed, responding only minimally to any stimuli. Although there was electrocardiographic evidence of myocardial damage and X-ray evidence of aortic calcification, electric shock therapy was started at the urgent request of her family, who desperately wanted something done to help her. After the second treatment, she opened her eyes and said a few words, for the first time in five years. After the third treatment, she kept her eyes open and talked incessantly, describing dramatically her frightening visual hallucinations. She saw rats and snakes coming after her, people threatening her, and other terrifying visions, which she had tried to shut out of her consciousness before by keeping her eyes shut. After the fifth treatment, she was calm, well-oriented, and rational, able to converse intelligently with those about her, no longer hallucinating. She was given six electric treatments in the course of 10 days, and was discharged from the hospital in good condition. She did well at home for several months after her last treatment, but eventually relapsed when a sudden illness of her husband subjected her to more domestic responsibility than she could tolerate.

D) *Complications:* There were no physical complications arising from any of these electroshock treatments given at St. Joseph's Hospital. The most frequent complications seen elsewhere (1) have been fractures, and the most serious have been cardiac or respiratory arrest. Fortunately, nothing of this sort occurred in this series.

The only complications occurring in this group of cases treated at St. Joseph's Hospital were of a mental sort that would not be regarded as complications at all in a mental hospital, but which caused difficulties in controlling the patient in a general hospital, as shown by the following cases.

Case 10: J.V., a 56-year-old carpenter, was sent to the hospital by his family physician because of headaches and dizziness that prevented him from continuing his work. When seen in psychiatric consultation, he also complained of nervousness, depression, inability to sleep, and inability to relax. These symptoms had arisen in a setting of friction with his wife at home, worry about financial losses in his work, the recent death of a daughter, and a recent automobile accident. He had had occasional alternating episodes of depression and elation in the past. Because of his profound depression, with its accompanying danger of suicide, he was started on electric shock treatments. After the first two treatments, he became very cheerful, and his attitude changed from that of a melancholy introvert to that of an exuberant extrovert. He made sexual advances to nurses, prowled the hospital corridors with an over-friendly approach to every one he saw, and became difficult for the nursing staff of a general hospital to control. Electric shock treatments were continued until he had had six treatments in the course of two weeks. He left the hospital considerably improved, with his depression entirely gone, and the subsequent manic state fairly well controlled. He did well at home for a month after leaving the hospital, and then began to react to his domestic and business worries with a recurrence of uncontrolled manic behavior. For treatment of this manic psychosis, he was sent to a mental hospital, as manic disturbances are too difficult to control either at home or in a general hospital.

Case 11: H.B., a 41-year-old widowed nurse, was admitted to the Neurosurgical Service because of a head injury in an automobile accident. She complained of severe headache, and the enforced inactivity of bed-rest gave her too much time to brood about the recent death of her only son, and about her own insecure prospects for the future. She became very depressed, unable to eat or sleep well, crying most of the time. In an effort to control her depression and agitation, she was started on electroshock therapy, as she proved very resistant to psychotherapy. She was given five electric shock treatments in the course of 10 days, which cleared up her depression effectively. As her depression lifted, however, her aggressive, resentful underlying personality became more apparent, and she became more uncooperative and difficult to control in a general hospital. She began to accuse the hospital nurses of being cruel to her, she refused to stay in bed, she refused to eat and then shouted that she was being starved. Because of the difficulty in controlling her uncooperative behavior on the ward of a general hospital, she was transferred to a State Hospital, where she was given more electric shock treatments and made a satisfactory recovery in the course of several months.

Comment: While electroshock treatment is probably given elsewhere in general hospitals, only one other mention of it is found in the literature (2). The eleven cases reported above give a good picture of the results of prompt, vigorous psychiatric treatment in a general hospital, illustrating particularly how depressions can be relieved by electroshock therapy. In the cases treated in this way at St. Joseph's Hospital during the last few years, no complications occurred, except for the two cases cited where some difficulty in controlling the patient's uncooperative behavior was encountered. No special facilities were required in handling these cases, except for the psychiatrist's portable equipment and the attention of one nurse during the treatment and the half-hour interval of recovery afterward. Seldom was more than two weeks in the hospital necessary. The availability of such treatment meant that a short period of hospitalization re-

lieved disabling symptoms that otherwise would have persisted for months or even years. The patients were saved from the stigma and expense of a mental hospital commitment, and also from the difficulties of home treatment with its strain on their families and its danger of suicide. Probably no group of patients is more grateful to the hospital and physician than the depressed patients restored to health in this way. Providing facilities for this kind of treatment in general hospitals appears to fill a real need.

Summary: The place of psychiatric treatment in a general hospital is discussed, particularly in regard to electroshock therapy, with cases illustrating its effectiveness in treating depressions.

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SYMPOSIA ON NEOPLASTIC DISEASES

The Montgomery County Medical Society, the County Chapter of the American Cancer Society and the National Cancer Institute will present Symposia on Neoplastic Diseases in the main auditorium of the Clinical Center, National Institutes of Health, Bethesda, Maryland. The dates: July 16th and August 20th. The time: 8:30 P.M. Latest trends in cancer and current research will be discussed.

You are invited to attend.

Scientific Papers

THE HYPOCHLOREMIC, HYPONATREMIC PHASE OF LOWER NEPHRON NEPHROSIS

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"It is not sufficiently considered that men require more often to be reminded than to be informed."—S. Johnson

With the possible exception of Lucke, who introduced it in 1946, clinicians and pathologists agree that the term lower nephron nephrosis is misleading, in that morphological changes are not infrequently noted in the "upper nephron" as well. However, if one realizes that the term is not one of exclusion but rather implies the site of the predominantly demonstrable lesion, then it does not appear unduly objectionable. In addition seniority has solidly entrenched the phrase in medical nomenclature and recent attempts to dislodge it have only produced a nine headed hydra. It is therefore doubtful if lower nephron nephrosis will be replaced in the foreseeable future by the more general terms of "acute renal insufficiency," "acute renal failure," or "acute renal shut down," by the redundant "acute tubular necrosis" (1), or by the overly restricted "distal tubular necrosis" (2).

The causes of lower nephron nephrosis are legion but may, in general, be divided into vascular and toxic classes. Without concern as to relative incidence, prognosis, or completeness, several specific causes are listed as follows: crush injury, intravascular hemolysis, including incompatible blood transfusions, pregnancy, burns, hepato-renal syndrome, severe electrolyte imbalance, and poisons, notably carbon tetrachloride.

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The syndrome can be divided into four stages, each because of its distinctive features actually representing a syndrome within a syndrome. The first stage begins with the body's immediate response to the challenge by the specific cause and commonly consists of circulatory insufficiency of variable degree, rarely lasting more than twenty-four hours in duration. It is quickly followed by or subtly blends with the second stage of oliguria, variably progressing to anuria and uremia.

The mechanisms involved have not been entirely elucidated but explanations generally favor one or both of the following possibilities: 1) Due to renal ischemia or direct action of toxic substances, tubular epithelium reabsorbs all solutes indiscriminately rather than selectively. 2) The tubules are blocked by pigment casts, thus preventing satisfactory excretion of urine. Renal interstitial edema and renal shunts have also been advanced as basic mechanisms but these appear to be on more tenuous grounds than the above.

The oliguric stage has been publicized so universally that few attending physicians would subject the patient to the injudicious administration of parenteral fluids. Although hyponatremia and hypochloremia are possible complications, these are usually of minimal degree and readily corrected by supplying modest amounts of sodium chloride. Hyperkalemia is apt to be more troublesome and may require the institution of simple measures such as restriction of potassium intake, the use of non-potassium containing resins, digitalization, parenteral glucose solutions with insulin, and intestinal intubation.

Adrenal cortical hormones are contraindicated. When markedly elevated levels of potassium threaten disintegration of the electrocardiogram, use of the artificial kidney is the only certain method of restoring serum potassium to normal.

After 7 to 10 days, the third or diuretic stage ensues. On occasion this has been labelled the convalescent phase but nothing could be more mis-described. As the mythical siren nymphs lured the unwary but willing seafarer to his destruction, the onset of voluminous diuresis and the rapid disappearance of uremic stigmata may seduce the attending physician into a state of complacency. With the kidney excreting an isosthenuric filtrate due to tubular failure in reabsorbing electrolytes, critical depletion of sodium and chloride, and often of potassium, magnesium, and phosphates, rapidly develops. If sufficient wastage occurs, this may ironically provoke a "second" lower nephron nephrosis on its own demerits.

This possibility must be recognized and appreciated. Daily measurements of serum electrolytes and salt intake are essential, and the daily quantitative determination of urinary chloride and/or sodium is desirable. Due to extreme wastage, extraordinarily large amounts of salt must be administered orally or parenterally to maintain normal or near normal serum concentrations of sodium and chloride. This stage usually lasts from one to two weeks but may persist for periods up to two months (3).

The fourth stage is characterized by the absence of clinical signs and symptoms but waning evidence of the disease is usually revealed by mild impairment of renal function and pathological abnormalities on renal biopsy. The precise duration of this stage varies from individual to individual but probably lasts several months.

Case Resume

A 50 year old white female patient was admitted to the St. Joseph's Hospital with a chief complaint of vaginal discharge of 3 months duration. There was no history of previous renal disease and no history of previous blood transfusions.

Physical examination: Patient was a well developed and moderately obese white female with a blood pressure of 218/100 and a pulse of 74. There was reddening and enlargment of the palatine tonsils. Lungs were clear to percussion and auscultation. Moderate enlargment of the heart was noted on percussion. A grade two blowing systolic murmur audible at the apex was referred to the other valvular areas. The second pulmonic heart sound was accentuated but intact. Abdomen was obese and soft. Liver, spleen and kidneys were not enlarged. An ill-defined, tender, movable mass was felt in the right lower quadrant. Pelvic examination demonstrated an enlarged retroverted fundus uteri with a mass attached to its left aspect. The physical examination revealed no other pertinent abnormalities.

Admission laboratory examinations: Hematocrit, 34 per cent; hemoglobin, 75 per cent; leukocytes, 9,050 cells per cu. mm.; normal differential count. Urinalysis, sp. gr. 1.022, negative albumin and sugar, occ. white blood cell; blood sugar, 98 mg. per cent; blood urea nitrogen, 9 mg. per cent; phenolsulphonphthalein 88 per cent excretion during the two hours following injection. EKG revealed evidence of coronary artery disease.

X-Ray examinations: Chest and intravenous pyelogram x-rays were interpreted as being within normal limits.

Course in hospital: On the eleventh hospital day, the patient underwent a dilatation and curettage, subtotal hysterectomy, and bilateral salpingo-oophorectomy under general anesthesia for myomata uteri and endometriosis. The blood pressure which was 140/80 just prior to surgery dropped to 80/50 during the dilatation and curettage. Vasopressor agents and whole blood were administered, with the pressure restabilizing between 180/100 and 150/80. During the immediate post-operative period, it was noted that the patient was oliguric despite adequate

parenteral fluids. A urine specimen was grossly bloody in appearance. Bilateral ureteral catheterizations ruled out post-renal obstruction. It was then learned that through a blood typing error, although she was type O Rh positive, she had received 600 ml. of type A Rh negative blood during her operation. She became icteric and developed a total serum bilirubin of 3.3 mg. per cent.

With the limited administration of parenteral fluids, the urinary output gradually increased and the blood pressure remained at 160/90. However, the patients clinical appearance correlated more closely with the elevated blood urea nitrogen and electrolyte imbalance. On the 7th post-operative day, she had emerged from the oliguric state, and three days later critically low levels of chloride and sodium were coincident with the onset of the diuretic phase. Her relatively insignificant salt intake was supplemented accordingly with 14.5 to 21.5 grams of sodium chloride daily. This amounted to 250–360 m/Eq/l of sodium per day and eventually proved adequate in restoring the deficit to normal. The serum potassium concentration which had previously attained a level of 8.3 m/Eq/l per liter dropped to normal. Carbon dioxide remained low throughout this period. Variations of the blood urea nitrogen, other serum electrolytes, fluid intake and urine output are tabulated in figure 1.

On the 16th post-operative day, an elevated temperature was present without other notable changes. Escherichia coli was cultured from the urine. When there was no response to tetracycline, intramuscular penicillin was added to the antibiotic regimen. Nevertheless, the patient went downhill and despite heroic measures expired on the 19th post-operative day.

We are indebted to Dr. William Lovitt of the Medical Examiner's Office for the following postmortem findings: Grossly arteriosclerotic narrowing of the aorta; moderate left ventricular enlargment of the heart with areas of fibrosis and marked coronary artery sclerosis; empyema,

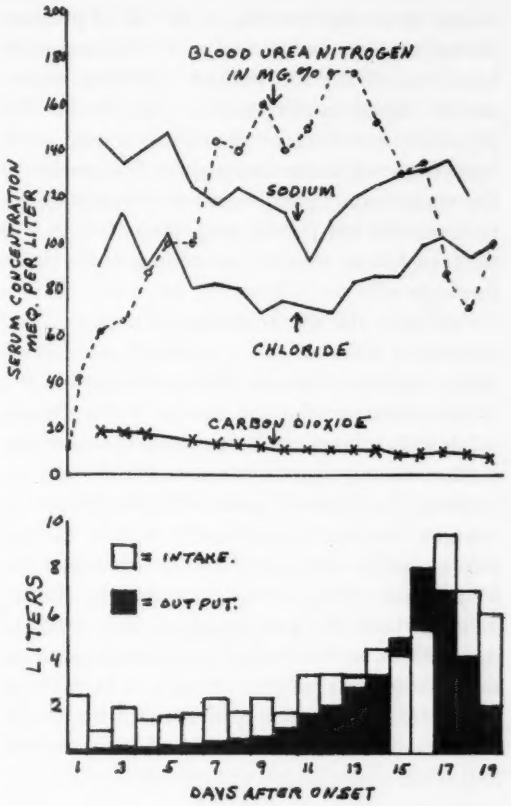

FIG. 1

left; bilateral bronchopneumonia, probably secondary to septicemia; acute splenic tumor; hepatomegaly; and bilateral pyelonephritis. Hemolytic Staphylococcus aureus, Proteus mirabilis, and Aerobacter aerogenes were cultured from the kidney although the latter two organisms probably represented contaminants. Microscopic examination of the kidneys demonstrated residuals of lower nephron nephrosis including persistence of pigment casts in the distal tubules but the major changes were due to a severe acute pyelonephritis. There was no evidence of nephrosclerosis.

DISCUSSION

The patient's lower nephron nephrosis probably resulted from two independent but additive

causes: first, the lowering of the blood pressure during surgery, and secondly, the intravascular hemolysis. With the patient receiving incompatible blood under general anesthesia, the physician was divested of any advantage stemming from subjective complaints. The nature of the transfusion error precluded its ready detection and did not permit early discontinuance of the transfusion, conceivably adding to the situation's gravity.

Only with the administration of large oral and parenteral doses of salt (14.5–21.5 gms. daily) was it possible to restore the serum chloride and sodium concentrations to normal levels. In our opinion the diuretic salt wastage phase is as important as the oliguric stage but with rare exception, has not been sufficiently emphasized in the past. Not only must there be massive sodium chloride intake but its continued administration for sustained periods may be necessary. Hunter and Muirhead (3), who have been quite aware of the problem, have reported two instances wherein salt wastage was present for 60 and 44 days respectively. During this period urinary chloride excretion varied between 350 and 800 meq. per day. This corresponded to an output of 20 to 48 grams of sodium chloride daily.

Although it was felt the patient was making a satisfactory recovery, the appearance of an acute fulminating infection was regrettable. The terminal pyelonephritis and bronchopneumonia due to a penicillin resistant Staphyloccocus confirms the alarm recently expressed in the literature. It also perhaps points to increased susceptibility of these patients to intercurrent infection which has been so troublesome and frequently fatal in other varieties of nephrosis.

SUMMARY

A case of lower nephron nephrosis secondary to renal ischemia and intravascular hemolysis is presented. The clinical stages, their causes and mechanisms are discussed. The importance of the diuretic phase with its tendency toward hyponatremia, hypochloremia, and possible increased susceptibility to intercurrent infection are emphasized.

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DUPLICATIONS OF THE ALIMENTARY TRACT

A Report of Two Cases

A. ANDREW ALECCE, M.D. AND WILLIAM A. RODGERS, M.D.

Duplications of the alimentary tract have been recorded in the literature by many different names until the above terminology was popularized by Ladd and Gross. Some of the synonyms used to describe this condition are "mesenteric cyst," "enterogenous cyst," "enteric cyst," "giant diverticula," ileum duplex," "gastric thoracic cyst," and "reduplication of the intestine." The rarity of this condition is evidenced by the number of single case reports in the literature, although several large series have been recorded recently.

We wish to thank Dr. John W. Ashworth and Dr. John J. Gould for allowing us to use their patients in this report.

Such anomalies may be defined as spherical or tubular structures intimately related to normal

bowel whose walls contain smooth muscle are lined by mucous membrane resembling some portion of the alimentary tract. This mucosa is not necessarily similar to the type possessed by the adjacent bowel.

They may occur from the base of the tongue to the anus, but the greatest number are related to the small intestine where the ileum is favored. There is a remarkable degree of variation in size and shape of the duplications, the tubular ones resembling segments of intestine for varying lengths, while others may be diverticular or spherical in type, at times attaining a size larger than a grapefruit. These may or may not communicate with the bowel.

If the duplication contains fluid, it is usually a thick, clear, colorless substance unless local pressure or necrosis has discolored it to a bloody or "muddy" shade. Normal intestinal contents may be present if an adequate communication exists with the adjacent bowel.

Although duplications have on occasion been referred to as "mesenteric cysts," it is important that they be differentiated from them. The true mesenteric cyst has a very thin wall; it is of lymphatic origin, and can be easily dissected free. A duplication has a thicker muscular wall and it is intimately adherent to the adjacent viscera. It often shares a common wall with the neighboring bowel, and can be removed only by resecting a portion of the local intestine.

Embryology

The classical work of Lewis and Thyng[5] is referred to often for evidence supporting the theory that duplications are the end result of fetal diverticula. These investigators found that in the pig, rabbit, and human embryo, small nodules of intestinal epithelial cells occurred regularly, sometimes degenerating into cysts, and that such structures could give rise to pathological diverticula if they became pinched off. Webster[10], however, drew attention to the fact that these pouches in the endoderm of the embryonic intestine are located on the outer mesenteric border, much at variance with the frequency with which duplications are located on the mesenteric surface of the bowel.

Bremer[3] clearly distinguishes congenital diverticula from duplications. True duplications, he stated, originate "by an abnormal persistence of the vacuoles normally present among the massed cells of the 'solid stage' of the intestine, a phenomenon of growth in embryos of the sixth or seventh week. By the confluence of a chain of vacuoles, a new channel is formed, parallel to the original lumen."

Menten and Denny[7] feel that a more adequate explanation is offered by the "organizer" concept of morphogenesis. They suggested that extraneous factors may "act on the human embryo to modify the normal formative 'organizer' effect so that developing plastic tissue may undergo pathologic differentiation." This would seem to provide a more adequate explanation of doubling of the genito-urinary system and lumbosacral spine that is occasionally associated with doubling of the colon. Ravitch[8] expresses similar sentiments when he calls this a "partial twinning."

Incidence

The greater majority of duplications are found in early childhood, especially during the first year of life, with only an occasional case occurring in late adulthood.

Signs and Symptoms

The symptoms that these anomalies produce are quite diverse because of the variations in size, shape, and location. Gastrointestinal hemorrhage, obstruction, and/or pain are the most common manifestations. If the duplication locates itself in the thorax, hemoptysis, hematemesis, or symptoms of pulmonary compression may be expected. Those occurring further along the gastrointestinal tract will produce symptoms peculiar to their location and oftentimes may be palpable as a round, smooth, freely movable mass. Roentgenologic investigation is usually of

great assistance in delineating the size, shape and location of the duplication.

Treatment

The only method of treatment for this anomaly is surgical and resection of the duplication with its contiguous bowel is not only preferred but usually necessary. Other choices include partial excision, marsupialization, and removal of part of the common wall shared by the duplication and adjacent gut. The general condition of the patient is an important factor in deciding the method of treatment, since it may be necessary to resort to an exteriorization and Mikulicz resection in a poor-risk infant.

Patient Reports

1. J.R.P., an eleven day old white female was admitted to St. Joseph's Hospital on September 28, 1949, with a diagnosis of intussusception. The child had been delivered at term at another hospital after an uneventful pregnancy. She was well until the evening prior to admission when she cried incessantly for five hours. This was followed by a rather large bowel movement containing a small amount of blood. Her family physician, noting a mass in the child's abdomen, sent her to the hospital, suspecting an intussusception.

When examined she appeared well nourished, weighing eight pounds, and was sleeping quietly. Her rectal temperature was 99.2 degrees Fahrenheit. The head, neck, heart and lungs were normal. A smooth, round, cystic mass the size of a hen's egg was palpable to the left of the umbilicus. This could be displaced laterally or medially for a short distance with no evidence of pain or tenderness. Peristaltic activity was normal, with no abdominal distention. The remainder of the physical examination revealed no abnormalities. An impression of Wilm's tumor or reduplication cyst of the intestine was made at that time.

Laboratory Data: Complete Blood Count: Hemoglobin 106%; red blood cell count 5,070,000 per cubic mm.; white blood cell count 12,500 per cubic mm.; normal differential.

Urine: Trace of albumin, and 2 to 4 red blood cells per high power field.

Stool: Positive for occult blood.

A barium enema examination revealed a mass in the abdomen which appeared to lie posterior to the colon, "probably representing a large left kidney."

On October 5, 1949, the child underwent laparotomy, and approximately thirty-five centimeters proximal to the ileo-cecal junction there was found a cystic mass five centimeters in diameter lying in the mesentery and intimately adherent to the wall of the ileum. In an attempt to separate the cyst from the ileum, it was found to possess several layers of muscle. The two structures were so firmly adherent that it was impossible to separate them without injuring the bowel. The cyst was opened and by pressing the opposite wall of the mesentery, it was turned inside out. It possessed a lining of mucosa and contained clear mucus under tension. It did not communicate with the ileum. In order to preserve the blood supply to the bowel, and at the same time forego an intestinal resection, the lining membrane was "peeled" from the cyst cavity and the remaining muscular walls approximated with cat-gut sutures to obliterate the "dead space." The abdomen was closed in layers without drainage.

The post-operative course was uneventful. The child took its formula well, and was discharged on October 16, 1949, weighing eight and a half pounds.

The pathological diagnosis was "congenital anomaly, duplication cyst of the ileum."

2. C.E.L. is a nine months old white male twin who was first admitted to St. Joseph's Hospital on July 10, 1946, because of vomiting. He was a premature weighing four pounds, five ounces at birth. At three weeks of age he started to vomit after each meal and failed to gain

weight. The vomiting gradually subsided until the day of admission, when he again vomited a large amount of dark material.

Physical examination revealed an undernourished, poorly developed male child weighing 12.5 pounds. His rectal temperature was 103° F. with a pulse of 200 per minute. A 5 cm. tender mass was palpable in the right upper quadrant of the abdomen. It was freely movable and separate from the liver. The remainder of the examination was negative except for a reducible right inguinal hernia. Stool examinations were positive for occult blood. A gastrointestinal series revealed a mass pressing on the pylorus with some retention of barium in the stomach after six hours.

The infant was given parenteral glucose and saline with vitamins, and a small blood transfusion in preparation for surgery. On the seventh day the mass suddenly disappeared and the temperature returned to normal. He simultaneously passed a tarry stool. Since the child continued to improve, surgery was cancelled.

It was decided that this was either a cyst that had evacuated its contents into the lumen of the bowel or an intussusception which had successfully reduced itself.

At 20 months of age he was readmitted for repair of the right inguinal hernia, which had become quite large. At that time he was physically well and there was no palpable abdominal mass.

He remained well until Feb. 9, 1951, when he was readmitted at the age of 5 years, with upper abdominal pain, nausea and constipation. On the morning before admission the parents again noticed the mass in the upper abdomen. The physical examination revealed an acutely ill, well developed child with a temperature of 99° F. and a pulse rate of 104 per minute. There was a slightly tender, firm, cylindrical, immobile mass in the right upper quadrant of the abdomen. There were no other pertinent physical findings.

Laboratory data: Hemoglobin 79%; red blood cell count 4,020,000; white blood cell count 19,150 per cubic millimeter with 80% filamented polys, 4% non-filamented polys, 15% lymphocytes, and 1% monocytes. Stools were negative for occult blood. A diagnosis of intussusception was made and an emergency laparotomy performed. A 16 cm. gangrenous cyst was found in the right upper quadrant. It was firmly adherent to the first and second portions of the duodenum.

Enucleation of the cyst was impossible. It was opened and a large quantity of foul-smelling pus evacuated. The cyst wall was excised, the defect in the wall of the duodenum repaired, and an anterior gastroenterostomy completed the operation. The child made an uneventful recovery and has remained well until the present time.

Microscopic pathology revealed a duplication of the duodenum with marked inflammatory changes in all of the layers of the resected cyst.

Comment

Patient No. 1 represents an unusual method for partially resecting a duplication cyst. Although second best to a total resection, it may be indicated where a limited but adequate amount of surgery is contemplated, especially in those instances where the cyst possesses no communication with the intestine.

Patient No. 2 is a classical example of a duplication of the duodenum which communicates with the intestinal lumen. The cyst obviously evacuated its contents into the duodenum during the first admission which accounted for the disappearance of the mass and cessation of symptoms. The child remained symptom-free until inflammatory changes finally obliterated the communication and the cyst refilled becoming symptomatic again.

Summary

1. Two patients with duplication of the alimentary tract are reported.

2. An unusual method of treatment is suggested in the first patient.

3. The second patient presentation stresses the long interval between acute episodes because of spontaneous evacuation of the cyst contents into the intestinal lumen.

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A PRACTICAL APPROACH TO THE TREATMENT AND DIAGNOSIS OF GASTRO-INTESTINAL HEMORRHAGE

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I had the pleasure about a year ago to present before the Medical Division of the Veterans Administration, Baltimore Regional Office, a basic working formula for dealing with gastro-intestinal hemorrhage. These thoughts were published in Clinical Medicine to which credit is given for the material included in this review. Much is added, however, in modification and clarification.

In defining the subject for this essay, I would like to make it clear that I am discussing massive upper gastro-intestinal hemorrhage, sometimes referred to as critical hemorrhage. It is characterized by severe hematemesis and melena, sometimes one or the other or both together. Perhaps there is no better time to speak of this subject than this year since there have been so many hemorrhages during this past fall, and it seems to me, as it has to many others, that each time a severe massive upper gastro-intestinal hemorrhage appears in the hospital, our reaction is one of uncertainty. By this I do not mean that we have no standard operating procedure to guide us in dealing with these critical hemorrhages but that it appears we are not only somewhat distressed about definitive diagnosis but also about applying emergency treatment. It is for these reasons that I thought it best to try to bring into focus our present thoughts on the subject of massive gastro-intestinal hemorrhage.

Before doing so, however, I should like to allude very briefly to some patients seen recently on the wards at various hospitals. For example,

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there was a youngster of 12 who bled very profusely and who, subsequently, was found to have a duodenal ulcer. It was the impression of most of the physicians who saw this patient that such hemorrhage in youngsters was uncommon, but according to Ivy, one may look for this not infrequently. The real difficulty in these patients is that we do not x-ray them and therefore do not make a definitive diagnosis.

There was also a patient on one of the wards in the hospital, a woman, who came in for her second hemorrhage. It was severe, as was the first one. The medical man consulted the surgeon who hesitated about operating. The patient was x-rayed. The x-ray was negative. I was asked to gastroscope her and I found only a gastritis. Because she wished it and because the doctor in charge thought it advisable, she was operated upon. At operation, the surgeon opened her stomach, looked everywhere but could find no bleeding area. However, a subtotal resection was done. On that day, it happened that I was making Service Rounds and so, two hours after the operation, we went to Pathology and looked at her stomach which was then fixed in formalin. There, unmistakably, was an ulcer about the size of a penny. This makes us wonder how such an ulcer could be missed by the various modes of examination. She, undoubtedly, would have been listed as massive hemorrhage, cause undetermined, except for her operation.

Then there was the Service patient who had received many transfusions and whom the surgeons were asked to examine. They felt that they would operate when his condition was safe but he never reached that point for after 27 transfusions which more than doubly replaced his circulation, he died. At postmortem, a very competent pathologist was unable to find the source of bleeding.

I mention these cases to show how difficult can be the quest for diagnosis in some of these bleeding patients.

To return to the subject of uncertainty as to diagnosis and treatment, when a patient appears in the hospital with a severe hemorrhage, the question always arises as to whether emergency diagnosis or emergency treatment is more important. In my judgment, emergency treatment is more important and this treatment comes down, at least for purposes of this discussion, to the administration of blood.

Many years ago in the days of Hurst, I should say prior to 1930, very few transfusions were given to bleeding patients. It was felt that they would stop bleeding as their blood pressures fell and as the coagulability factors came into play. Then we passed into the era when transfusions, administered promptly, were considered lifesaving and so it was only a question of how to measure the optimum number of transfusions a patient should get. Now recently, there has been a five year survey (which really means that ten years have passed in order to get a reliable five year survey) in which men like Pollard and Ingegno and, perhaps, Palmer feel that the results with multiple transfusions are not as good as has been reputed.

I am one who believes that a patient with massive hemorrhage should get 1000 cc. of blood and that this could be extended to 1500 or 2000 cc. but when one gets beyond that, he is approaching an exchange transfusion. This not only is hazardous because of transfusion reactions but because many other unmeasurable and undesirable things may happen. For example, Rienhoff, in a recent editorial in the Southern Medical Journal, has reported that platelets are depressed and that these patients develop ecchymosis. Perhaps there are other serious matters which we have not recognized, all secondary to multiple transfusions.

How, then can we decide how many transfusions a patient needs? I believe simple measurements such as anyone of us can do, consisting of hemoglobin, hematocrit, red cell count, blood pressure, pulse rate, and perhaps urea nitrogen on the blood are adequate. Now, I am well aware that blood pressures can change rapidly and that the patient can go into shock. I am also

aware that pulse rates can change rapidly. All of us know that hemoglobin, hematocrit, and red blood cell counts do not follow quickly the loss of blood and in many cases, perhaps in most of them, these measurements will be very high. These may mislead the observer, making him believe that the patient is in good condition only to find a few hours later, when hydremia sets in, that the patient is critically anemic. But, if all these five factors are followed simultaneously and frequently and since they are easily measurable and done accurately by any capable person, they will form a satisfactory guide as to the patient's progress and as to the need for blood. It was formerly considered and still is valid that a pulse rate which rises, say from 80 to 90 to 100 to 110 or 120 shows that the patient's hemorrhage is not under control, and also that a blood pressure which falls from 130 to 100 and perhaps 90 or 80 indicates that this patient is losing ground. When the pulse rate is going up and the blood pressure is coming down, then, of course, this patient may no longer be a medical problem and conservative measures must be abandoned. Blood is not controlling the situation and surgery should be considered. At the same time, the other measurements of blood such as the hematocrit, hemoglobin and red cell count will also be following this trend. Now when all these factors are stabilized, one will feel that the patient's condition is under control but only when the pulse rate is decreased, the blood pressure goes up, and the factors measuring the amount of blood are all on the upward trend, can we be sure that the patient is really out of danger and that the hemorrhage has ceased. It should be remembered, too, that all this must take place in a prescribed period of time. It has been agreed by most observers that 48 hours is a reasonable time for a severe hemorrhage to be brought under control. If it is not controlled in that period of time, then the patient is in fact, no longer medical and becomes a surgical problem. If the period is prolonged to 72 hours and longer, then the best time for operation may have been lost.

There are other factors to be considered in deciding whether a patient is medical or surgical. For example, the age factor. If the patient is older, above 40, vessels are arteriosclerotic or beginning to be in that category, then perhaps one would not gamble with a hemorrhage which seems to be continuing. If a hemorrhage is brought under control, only to recur, then that patient may be surgical. If a hemorrhage is stabilized but not quite well enough, that patient may be surgical. If the bleeding is rapid, the patient may be surgical. In fact, one may consider, in giving blood, that he is not only trying to stabilize the patient but may be getting this patient into good condition for surgery.

There are many other factors which can measure the rate and quantity of loss of blood, in addition to the age factor, which will help us to decide in a reasonable period of time whether we should treat the patient medically or surgically. These observations then sum up briefly the present status of emergency treatment for massive gastro-intestinal hemorrhage.

Now, as to the question of diagnosis, there is a trend today toward early and immediate diagnosis. There are some clinics in which the patient in severe hemorrhage (unless he is in shock) will be esophagoscoped, gastroscoped and x-rayed within, perhaps, a two-hour period and it is said by some of these groups that the percentage of diagnosis and the percentage of better results justifies this approach. I am not one who belongs to this school of thought. I have always objected, for example, when the residents in the hospital will call excitedly and say that the patient is bleeding badly; will one come and do an esophagoscopy and gastroscopy and tell them whether the patient is bleeding from the esophagus or from the stomach, so that the surgeon will know where to operate. I concede that it is valuable to the surgeon to know where the bleeding is originating. I think it is desirable for all of us to know

this, but if one will analyze the situation, he is far better off than he believes. We know, for example, that 7 out of 10 massive upper gastro-intestinal hemorrhages occur from ulcer, and since duodenal ulcer is more common than gastric ulcer, it is the duodenal ulcer which accounts for most of the hemorrhages. If added to the diagnosis of ulcer, one considers gastric carcinoma, gastritis and esophageal varices, he can account for 90 per cent of the cases of bleeding. Now, if the surgeon says he would like to know where to operate, I would suggest that the incision for gastric carcinoma, for ulcer, and for gastritis is pretty much the same, the great difference being in the degree of operation. Therefore, it becomes important only to decide whether esophageal varices are present.

I also know that esophagoscopy may pick up esophagitis or esophageal varices, etc. and that this is all very helpful. Likewise, gastroscopy may disclose gastric erosions, gastric ulcer or hemorrhagic gastritis. The latter, incidentally, is more common in cirrhosis than we now think. I also know that early x-ray may disclose an ulcer or carcinoma. It is my impression, however, that a barrage of endoscopic procedures and x-ray are not routine in most clinics. First, there are not many people who can perform these procedures capably; also these procedures are not without their dangers; and on the whole, a good clinical approach may be just as valuable. For example, I think it should be routine in any massive gastro-intestinal hemorrhage not only to measure the degree of hemorrhage but also to do a few liver function tests, because it is unlikely that there will be liver damage when the cause of bleeding is due to hemorrhage from ulcer. It is also more likely that in the presence of liver damage, as demonstrated by liver function tests, varices will be present. I know that anywhere from 2 to 25 per cent of the cases of cirrhosis may be associated with ulcer but the very spread of this percentage rate introduces quite an uncertainty and makes it likely that a positive bromsulphalein test, for example, points to varices rather than ulcer and vice versa for a negative test.

Early x-ray is important and here I would like to say a few words about the historical development of this procedure. Some years ago, everyone did the Gastro-intestinal Series after about two weeks; in other words, a safe period after hemorrhage had ceased. In recent years, due to the introduction of the non-manipulatory method, these x-rays have been accomplished during the period of hemorrhage and in some areas, a greater percentage of positive x-rays has been the result. However, many radiologists prefer to do their x-rays with manipulation later on and only in a few clinics, do the x-ray men favor very early examinations. If they wish to do them early (and in the occasional case) I am all for it but as a routine procedure, I do not think it as valuable as it was originally thought to be. Shortly after hemorrhage ceases, I believe one can do an x-ray and find out what is going on. Sometimes an x-ray two and three months after cessation of bleeding is valuable because by this time the scar will indicate the area where the bleeding came from whereas prior studies may have been persistently negative.

Now with all this diagnostic effort, there is a group of 5 to 20 per cent of patients in whom the cause for bleeding cannot be determined. One of the reasons for introducing early diagnostic procedures was to decrease the number in this group. Of course, another reason was to locate the area of bleeding in case the surgeon should have to operate. If one takes time to talk to these patients, providing they are not too ill, and one gets an ulcer history, that is very helpful. If one gets a history of alcoholism, that, too, will be helpful in pointing to the possibility of esophageal varices. If one gets any history indicating carcinoma, such as an irregular story or a sudden onset of indigestion or some of the later symptoms of anemia and loss of weight, then he may think in terms of gastric carcinoma.

A physical examination during a period of hemorrhage should be limited. It should take into consideration the blood pressure, the pulse rate, whether the abdomen is rigid, whether there is a palpable liver, but an effort to do a very thorough examination is to be avoided since these patients should not be subjected to such an intense study. If this is to be avoided, then it seems to me that endoscopic and x-ray procedures should be considered very carefully before they are done routinely since they, too, can be traumatic.

Let us now say a word about surgery in these cases. If one knows there is a lesion, he can go in and eradicate it. That is fine definitive therapy. Suppose a lesion is not known and cannot be found, what should one do? I have, in the recent past, been converted to the blind subtotal gastrectomy. In a study of the literature and in my own private work, I feel sure that a subtotal gastrectomy is the least one can do in a bleeding patient when the cause is not known, but is presumed to be in the upper gastro-intestinal tract. If one reflects for a moment, the removal of part of the stomach removes the acid-bearing tissue and therefore, takes care of the ulcer tendency. If there is a carcinoma, certainly that much and more would be removed and even in esophageal varices, the removal of this vascular bed would reduce some varices or volume of blood flow, at least, during the period of emergency. I am no longer sympathetic to a surgeon who goes in, cannot find the area of bleeding and backs out. I am afraid such an approach is no longer to be recommended.

I should like to say a word about a classification of bleeding which I have mentioned on other occasions. There is a group of bleeders in whom the cause for the bleeding is known—let us say, a peptic ulcer. There is another group of bleeders in whom there is a cause of bleeding demonstrable, such as an ulcer, but at operation, another bleeding area is found. For example, in a recent patient, a leiomyosarcoma was the cause of bleeding and the ulcer was not bleeding at all. The question which arises in these cases is whether the non-bleeding area should also be removed. That is left to the judgment of the surgeon.

Then there are bleeders in whom a cause for the bleeding is found but it does not seem to be adequate. For example, in one of our recent patients, an atrophic gastritis was found but we did not feel that it was an adequate explanation of the bleeding. Finally, there is a group in whom no cause for bleeding can be found and here the question continually presents itself as to what is to be done. I have, as you know, for some time been suggesting that, in some people with hypertension, there may be vicarious bleeding from the gastro-intestinal tract. For these patients, in whom the cause of bleeding cannot be found and especially those controlled by medical measures, I believe that with repeated hospitalizations and examinations of every kind (including the occasional operation during the period of bleeding) the source of bleeding will eventually be disclosed in many of them.

I should like to discuss very briefly the bleeding which occurs from esophageal varices. The emergency treatment is tamponade which is the oldest method of treating a bleeding area that we know. Such tamponade is done by a tube such as Sendstaken has devised. Properly used, it may be lifesaving. There are surgeons, such as Linton, who do a transfixion of the bleeding esophageal varices. Others, however, do not believe this is a good operation. Definitive therapy for bleeding esophageal varices is the portacaval or splenorenal shunt; there is much which should be said on this subject. I believe today more and more surgeons are doing these operations very successfully. In the early days of this operation, Whipple and Blakemore did a non-suture splenorenal shunt, sacrificing the left kidney. Thrombosis was a problem and the mortality rate was considerable. Later, they and others (Linton, et al.) introduced the suture method without sacrificing the left kidney but removing the spleen, as is always done in splenorenal shunt. If the

spleen is removed and the vein is not made use of immediately in operation (the splenorenal shunt), then the time for using it is lost. It is to be emphasized that these operations are being done more and more frequently and successfully. For one thing, we know how to select our cases better. For example, a persistent hypoalbuminemia, hypoprothrombinemia, the presence of persistent ascites or persistent jaundice or advanced cirrhosis are contraindications to the operation. The operation, incidentally, is done primarily for bleeding and not for ascites nor for improving liver function although there is evidence that, in the more recent studies, liver function is being improved and there is reason to believe that some of the ascites cases are aided by this approach. Finally, I do not believe that tying off the hepatic artery or the splenic artery is the therapy of choice although they might be considered emergency therapy especially when the splenic artery is tied off.

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KARTAGENER'S SYNDROME

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Kartagener's Syndrome is a rare triad consisting of total situs inversus, bronchiectasis and rhinopharyngosinusopathy. A total of 104 cases of Kartagener's Syndrome had been reported up to 1955. The following case is presented in order to add a previously unrecorded case to medical literature. The case also emphasizes the need for critical evaluation of pulmonary parenchymal pathology occurring in the presence of acute abdominal disease.

Case Report

A 41 year old white male railroad brakeman was hospitalized on October 3, 1955 with the chief complaint of left upper quadrant abdominal pain, duration 24 hours.

On the day before admission, the patient developed cramping abdominal pain, worse on the left side. He worked that night and had a bowel movement at 4:00 A.M. On the morning after onset, the pain became worse and definitely localized to the left upper abdominal quadrant. The pain was made worse by cough, deep inspiration and turning. There was no nausea or vomiting and no change in bowel habits. Urination was free, although the urine may have been slightly lighter in color than normal.

On admission he had an immediate flat plate of the abdomen which was interpreted as follows: "Patient has a complete situs inversus. I believe there is some mottling at the base of the right lung which may represent an acute pneumonitis. No abnormal calculi are noted" (Fig. 1). (It was at this point in the patient's clinical course, due to the presence of total situs inversus and mottling at the base of the right lung, that the diagnosis of Kartagener's Syndrome was first entertained and entered on the clinical chart.) Other laboratory work on admission: Hemoglobin 103 per cent, Leucocytes 11,450; Polymorphonuclear leucocytes 80; Filos. 78; Non-Filos. 2; Lymphocytes 20. Serum Amylase 45 units.

The past history and family history are given in detail because of the apparent congenital nature of the components of the syndrome.

Past history revealed that the patient was born in Pennsylvania as a full term hospital delivery. The only known childhood disease was measles. The patient had one episode of pneu-

FIG. 1

monia in 1949 or 1950 and was treated at home for six weeks; during this illness, no x-rays were taken. The patient was inducted into the Army in 1937 and during his pre-induction physical examination was told that he had thoracic situs inversus. He served in the Military Service from 1937 to 1940 and was hospitalized for heat exhaustion.

Family History discloses that the patient's father was born in Russia near the Polish border and was about 27 years of age at the birth of the patient. The father died in 1940 in his sixties with or because of asthma. The mother was born in Russia as a Russian Pole and was about 25 years of age at the birth of the patient. She died in 1955 at age 67 with diabetes. There were three children and the patient is the second child. Both siblings are living and do not have situs inversus.

In the systemic review, questioning elicited the fact that the patient started to smoke cigarettes at age seven. He formerly smoked three packages a day and now smokes one package a day. The patient claims to have had sinusitis as far back as he can remember, with right sided nasal obstruction and a post nasal drip. There is early morning throat clearing and he has coughed so hard that vomiting has occurred. His cough is intermittent with no streaking of blood and no frank hemoptysis.

Following the initial investigation, the patient was ordered complete bed rest, clear liquids by mouth, fortified Crystacillin, 400,000 units intramuscularly and Demerol for pain. On the following day, October 4, x-ray of the chest showed the heart to be enlarged in transverse diameter with some preponderance of the left ventricular salient. Right lower lobe pneumonia was reported; the pleural spaces were clear.

Because of increased pain and difficulty in making a definitive diagnosis, laparotomy was performed on October 4, the day after admission, under general anesthesia. At operation, the typical findings of abdominal situs inversus were seen in that all abdominal organs were mirror images of the normal. The gallbladder was slightly thickened, changed in color and there were numerous pericholecystic adhesions. Exploration further disclosed a wedge-shaped omental infarct due to spontaneous torsion of

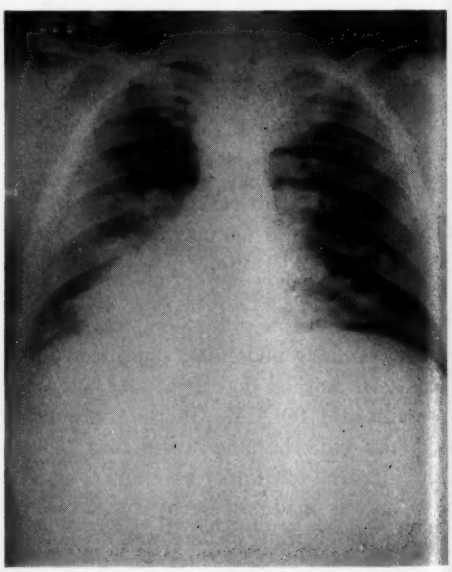

FIG. 2

the omentum and this was excised. Routine appendectomy was performed.

Post operatively, the patient was troubled by persistent cough. On October 10, 1955 x-ray of the chest revealed heavy infiltration in the lower portion of both lungs, suggesting bronchiectasis. No appreciable change had occurred since the previous examination of the chest (Fig. 2).

During the patient's post operative course, in order to complete the diagnosis of Kartagener's Syndrome, x-ray of the paranasal sinuses was taken and revealed mucosal thickening of all the sinuses apparently due to chronic infection, and complete agenesis of frontal sinuses (Figs. 3, 4). At this point, an ear, nose and throat consultation was requested of Dr. Bernard S. Kleiman. His report follows:

Ears: Right drum is retracted with some minimal calcium deposits on the periphera and minimal injection along the handle of the malleus. The left drum is thin with excellent landmarks except for the periphera which shows marked evidence of calcium deposits. The external canals are normal. Hearing appears basically normal.

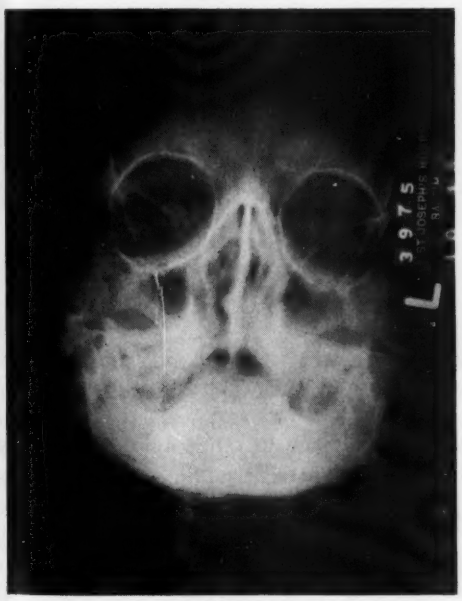

FIG. 3

FIG. 4

Nose: Generalized right septal deviation on which is superimposed a ridge paralleling the inferior turbinate and abutting against the inferior turbinate in the posterior third. The mucous membrane color is normal. There is excessive mucoid to mucopurulent secretion on the floor bilaterally; large polyp on the right originating in the anterior middle meatus.

Mouth: Complete upper plate; no lower teeth or plate. Tongue is normal. Tonsils are present, small in size (plus 2) with minimal injection of the anterior pillars. No cryptic debris can be expressed with pressure over the anterior pillars. Posterior pharyngeal wall is normal.

Nasopharynx: Non-inflamed minimal lymph tissue in the vault of the nasopharynx. The Eustachian tube, orifice and fossa of Rosenmuller are not involved.

Hypopharynx and Larynx: Normal laryngeal movements. Both vocal cords are chronically reddened.

Neck: No palpable glands or masses.

Sinus x-rays: There is agenesis of the frontal

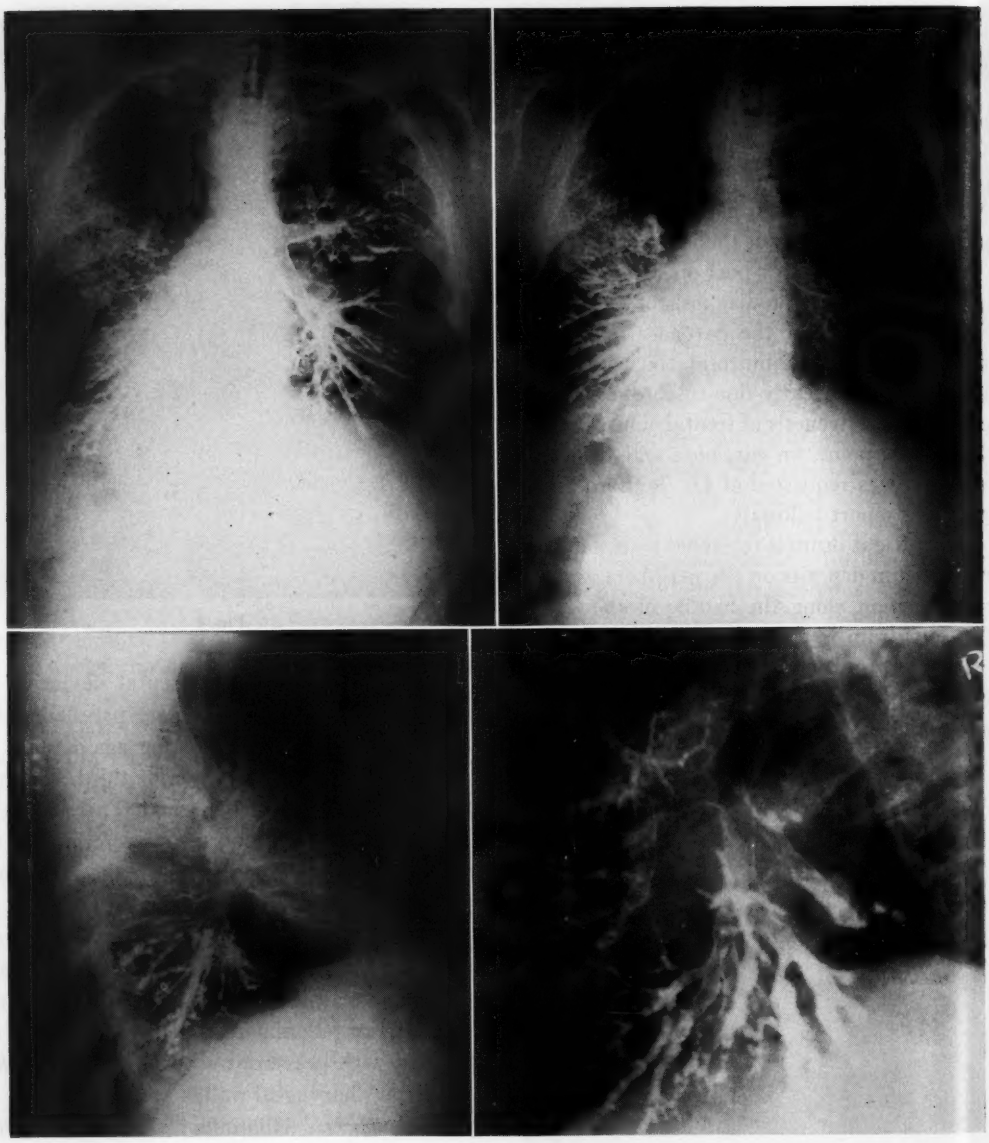

FIG. 5

sinuses. There is diffuse clouding of the right ethmoid and both maxillary sinuses.

Nasal Smear: Epithelial cells only; no eosinophils.

Dr. Kleiman's impressions were: 1) Bilateral chronic maxillary sinusitis and right ethmoiditis. 2) Nasal polyp, right. 3) Septal deviation, right. 4) Chronic laryngitis. 5) Agenesis, frontal sinuses.

The patient was discharged from the hospital on his eleventh post operative day with the final diagnoses of: 1) Kartagener's Syndrome. 2) Pri-

mary torsion of the omentum with omental strangulation. 3) Chronic non-calculous cholecystitis.

At a subsequent hospitalization on March 7, 1956, the patient had a bronchography which corroborated the clinical impression of bilateral bronchiectasis (Fig. 5).

Discussion

Kartagener's Syndrome is a rare triad of total situs inversus, bronchiectasis and rhinopharyngosinusopathy. Involvement of the upper respiratory tract includes deviation of the nasal septum and/or nasal polyposis; pharyngeal disease usually includes hyperplasia of lymphoid tissue; the sinusopathy may be inflammatory, degenerative or developmental in nature (1). The common developmental finding is hypoplasia, more often agenesis, of the frontal sinuses. Unexplained papilledema has occurred in one case (2), another patient has exhibited an anomalous left subclavian artery (3) and, in one reported case, there was a sister with a congenital heart of the Fallot type (4).

In 1887, George Fisher described total situs inversus and in 1904 Siewart reported the first case of Kartagener Syndrome in a 21 year old male (5). Oeri (1909) first described the autopsy findings in the triad (6). In 1923, Guenther reported Kartagener's Syndrome in a 19 year old female who had a sister with visceral transposition (7).

In 1933, Kartagener advanced the interpretation of a common congenital origin of the triad and etiologic correlation of the three components, and reported four cases (8). Two years later, in 1935, Kartagener and Horlacher published a total of 11 cases (9, 10). Other milestones in the literature of this syndrome include the paper of Adams and Churchill (1937) who added five new cases from the Massachusetts General Hospital (11), and the work of Olsen (1943), who added ten cases from the Mayo Clinic (12).

There were 104 cases of Kartagener's Syndrome reported up to 1955.

In Kartagener and Horlacher's series, bronchiectasis and sinusitis were associated with situs inversus in 20 per cent of cases.

Adams and Churchill, in analyzing 232, 112 patients from the Massachusetts General Hospital, found 23 cases of total situs inversus. The incidence of bronchiectasis and sinusitis in the total hospital admissions was 0.3 per cent (712 cases) whereas the incidence of bronchiectasis and sinusitis with situs inversus was 21.7 per cent (5 of 23 cases), or approximately 70 times the incidence of bronchiectasis and sinusitis in the general hospital admissions.

Olsen studied situs inversus during the years 1922 to 1941 at the Mayo Clinic and collected 85 cases. Bronchiectasis was present in 16.5 per cent (14 cases) and sinus disease in 10 of the 14 cases or 12 per cent. Only 0.5 per cent of the total number of admissions had bronchiectasis and sinusitis.

Adams and Churchill, in their scholarly presentation of the origin of Kartagener's Syndrome, suggested that two types of people are effected by transposition of viscera: 1) "Mutants"—situs inversus with no other abnormalities, and 2) "Monsters"—transposition of viscera associated with other congenital defects at various levels of the respiratory tract including bronchiectasis and faulty sinus development. As previously mentioned an anomaly of the left subclavian artery and congenital heart disease of the Fallot type have reported in association with Kartagener's Syndrome.

The theory that there is a common congenital origin of the three elements of the triad of Kartagener is further substantiated by the finding of Kartagener's Syndrome in twins and siblings (13–16). The bronchiectatic lesions, pathologically, are not different from those observed in the usual bronchiectasis of the so-called acquired type (17).

Comment

The presence of pulmonary parenchymal disease in a patient with signs and symptoms of

acute abdominal disease may indicate several possibilities; 1) The pulmonary disease may be the sole pathologic source of the patient's clinical picture; basal pneumonitis with diaphragmatic pleurisy, basal atelectasis, pleurodynia, etc. often confer the signs and symptoms of the acute abdomen; 2) The pulmonary disease may be entirely unrelated to the patient's abdominal complaints; concurrent pulmonary congestion, pneumonitis, tuberculosis, carcinoma, pneumoconiosis, etc. are coincidental accompaniments; 3) thirdly, the pulmonary pathology may arise as a result of intra-abdominal disease; pleural effusion, basal atelectasis and pneumonitis are caused by liver disease, such as cirrhosis and the infectious diseases of the liver (pyogenic and amoebic abscess, hydatid cyst, etc.), by acute cholecystitis and its perforative complications, by subphrenic infection and by peritonitis.

It was, therefore, of advantage to consider the diagnosis of Kartagener's Syndrome early in the course of this patient's disease. Awareness of the syndrome prevented uncertainty as to the nature of the pulmonary pathology in its relation to the clinical picture. Decisions concerning the need for surgery, the proper time for surgery and choice of anesthesia were thereby facilitated.

Summary

1) A case of Kartagener's Syndrome in a 41 year old male is presented.

2) Kartagener's Syndrome is reviewed; the three common components of the triad, situs inversus totalis, bronchiectasis and rhinopharyngosinusopathy are elaborated upon and the theory of their common congenital origin restated.

3) It is stressed that, when the physician is confronted with situs inversus, he must entertain the diagnosis of Kartagener's Syndrome and evaluate the nature of any associated pulmonary parenchymal disease in order to define its position with relation to the patient's symptomatology.

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WARTS, TYPES AND TREATMENT

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There are two principal types of warts, nevoid or neoplastic and infectious. The latter are due to virus infection. Under the former are put congenital verrucous epithelial hyperplasias and the seborrheic keratoses (also called seborrheic warts, senile verrucae, basal cell papillomas etc.).

Of the virus types, the most common are the verrucae vulgaris, which occur on the hands, face, feet and almost anywhere on the body. When they occur on the soles, they are called verrucae plantaris. Where the latter occur as multiple grouped warts they are called mosaic warts. The large verrucae which occur in warm, moist areas are called condylomata acuminatam and belong to the virus infectious group, as do also the smaller genital warts. It has not been proven that the verrucae planae juveniles are due to the same virus, and histologically they show a different and diagnostic pathologic picture.

FIG. 1. Senile wart (seborrheic keratosis) characteristic picture with a thick epidermis composed principally of basal-like cells with horn cysts.

Seborrheic keratoses are late nevoid lesions which are congenital and therefore predetermined before the patient is born, even though they may appear late in life. They are located principally on the torso, but may frequently occur on the face, especially the temporal regions and cheeks. They start as small light yellow to brown, fairly flat plaques and as they may grow larger in diameter, they also may become thicker and more elevated. As they take on the latter changes, they usually become darker, progressing from small yellow, flat papules to brown and even to dull black, large plaques, or even nodules. Some of these dull black lesions that have started early in life may be mistaken for malignant melanomas, but a biopsy should readily clear this misconception. The senile verrucae practically never undergo malignant changes.

HISTOLOGY OF WARTS

The seborrheic keratoses, which one of us prefers to call senile warts (except for the objection of using the term senile when talking to patients and from the histologic point of view are often called basal cell papillomas) are purely epidermal structures; that is, the changes start at the basal layer and extend upwards.

Seborrheic keratoses (Fig. 1) are composed of a confluent mass of basal-like cells in which there are usually many horn-like cysts. These cysts vary in extent, size, number and although the change from the basal architecture to the keratin is fairly abrupt, they nevertheless have a granular layer. There is usually no, or very little, parakeratosis in the horn cysts, unless the lesions have been irritated. These changes are in contradistinction to the histologic changes found in true pearls which occur in prickle cell carcino-

mas where the cells are of the prickle cell type and the change to keratinized cells is more gradual without the presence of a granular layer (because of the more abnormal keratinization in the carcinomas). Dyskeratosis in senile verrucas or evidence of malignancy are strikingly lacking. Dr. Frederick Weidman suggests that senile verrucas are some type of obsolete skin appendage, but he also noted that they do not occur in the higher anthropods.

The infectious warts (Fig. 2) are also purely epidermal structures histologically, but are composed mostly of prickle-like epithelial cells, except in the basal layer and above the keratohyalin layer where there is usually a variable, but usually increased, amount of keratin and parakeratosis. Hyperkeratosis, or horny material varies a great deal (from wart to wart). The characteristic histologic changes when present occur at the granular layer where one sees normal sized vacuolated cells with a dispersion of nuclear kerato-hyalin granules. Strauss and others have suggested that the more characteristic finding is the eosinophilic inclusion body in the cells at the granular layer. If the cellular, but especially the nuclear changes are not present in the granular layer, one may have difficulty differentiating at times the common wart from the premalignant warty type of senile keratoses.

Treatment

In the folklore (1) and literature (2–5) there are many remedies recommended for contagious warts, many of them claiming good results. In order to understand this problem, one has to refer to the natural history of the warts. Warts are much more readily cured in children than in adults. Memmerscheimer and Eisenlohr (6) followed about one hundred warts of several types for six months, half of which they treated with local application, the other half they simply watched. They found that the results were essentially the same and that 25 per cent of the common warts disappeared within six months, whereas 50 per cent of the verrucae planae juveniles disappeared in the same time.

The Psychotherapy of Warts

In 1934 Sulzberger and Wolf (7) presented an excellent summary of the treatment of warts by suggestion. They wrote that in the treatment of all virus warts psychotherapy plays a very important role. They quoted in detail Bloch's (8) original series with this form of therapy in which

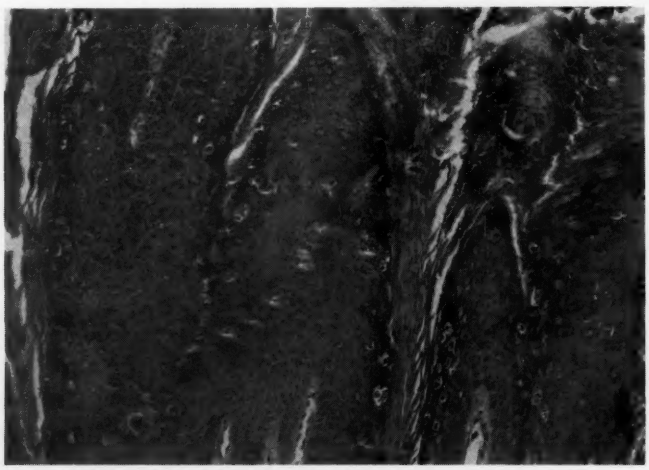

Fig. 2. Verruca vulgaris (common wart) with long pegs of hyperkeratosis. At the granular layer one can see the dispersion of the nuclear material in the vacuolated cell which is rather characteristic of the histologic picture of verruca vulgaris. The epithelial cells of the verruca vulgaris are more prickle cell in type than those in senile warts.

he reported a cure of 73.5 per cent of 179 patients followed. Sulzberger and Wolf did not consider in detail the psychotherapy in the treatment of warts comprising medical relaxation by hypnosis. This form of psychotherapy certainly should be more effective than the usual psychotherapy, but the average busy practitioner or dermatologist is not qualified to apply hypnosis because of the lack of training in this form of therapy. If warts can be cured by suggestion and if different types of therapy may be forms of suggestive therapy, then this may explain why certain investigators have more success with their own particular form of therapy than the others.

One of the well recommended methods of removing warts on the bottom of the feet, or plantar warts, is by roentgen-ray treatments (9). Again this works better in children than in adults, and also works much better if there are only one or two warts rather than many. The mosaic type usually does not respond to roentgen-ray treatments. A number of forms of treatments have been suggested within the last few years, including the curretting out of the warts, injections of novocaine under the warts, the ingestion of Vitamin "A," etc. In the authors' experience none of these methods have been satisfactory in the treatment of plantar warts. We have used euphorbium resin as recommended by Goldblum and Curtis (10) and Wright (11) in a number of patients, but have abandoned this method. Podophyllin (12–13) has been recommended highly for the treatment of venereal warts and condyloma acuminatum and works well if the warts are moist. One must be careful in applying podophyllin in order not to obtain too much reaction. Podophyllin tends to interfere with the development of the cells. When the warts are dry, this method is practically useless. It is in these cases that liquid nitrogen works well with the least discomfort to the patient.

There are many methods used for the removal of the late nevi (senile warts), among them being, of course, surgery and desiccation, but since they are benign epidermal structures, one can frequently simply currette them off and touch the base with an escharotic. They also respond well to radium and roentgen-ray treatments, but only in certain locations would this form of therapy be indicated. In fact, the removal of seborrheic warts is usually only indicated for cosmetic reasons. Occasionally in areas as under the breasts, axillas, etc., they may be subject to trauma and irritation, plus secondary infection.

Treatment by the Use of Liquid Nitrogen

A more recent method of therapy introduced for the treatment of these is the use of liquid nitrogen (14–16). Liquid nitrogen is a by-product of oxygen extraction from air. Liquid nitrogen boils at about minus 187 degrees centigrade. A fairly tightly wrapped wad of raw cotton, but larger than the usual amount, is put on the end of an applicator and saturated with the liquid nitrogen. It is usually preferable not to apply the liquid while it is still dripping from the cotton. The applicator with the liquid nitrogen on it is applied without pressure, by touching the tip of the saturated cotton to the growth being treated. The lesion will soon turn white and one should treat the lesion until a one or two mm. border about the lesion itself turns white. This shows that the tumor and the borders of the lesion have been frozen and a blister, or a potential blister will form between the lesion and the dermis. If a blister forms it should not be opened, but preferably the epidermis should be allowed to heal underneath the treated area and the treated lesion permitted to peel off. The patient is usually seen about every two to three weeks and if the wart has not fallen off, one may have to retreat the lesion. The advantage of this method is that although it is not painless, it is not very painful; the treated area usually develops no crusting, and healing is usually without scarring. The paronychial warts can be treated with liquid nitrogen without cutting

away the nail and the area usually heals without deforming the nail.

Verruca plana juveniles in children usually respond well to injection of bismuth, but in adults this form of wart may be more resistant to therapy. Liquid nitrogen is not too helpful since the warts are small and often numerous and it is difficult to give each wart sufficient contact with the agent. The treatment of plantar warts with liquid nitrogen is usually unsatisfactory, apparently because the keratin layer may be one centimeter thick and one must freeze below the basal layer of the epidermis to obtain results. This is very difficult to do because of the thick overlying keratin layer.

Summary

The two common types of warts are discussed. The nevoid type or the late congenital form which occurs in older people (usually above the age of thirty), is benign and the principal indication for their removal is cosmetic. Although they may be removed by different forms of therapy, liquid nitrogen is a very satisfactory method for their removal.

The other common form of wart is verruca vulgaris which is due to a virus infection. The various forms of treatment are discussed. Liquid nitrogen in this type of wart is a very satisfactory method of treatment, except for plantar warts.

Conservative measures in the treatment of warts are usually rewarded by satisfactory results, although in a moderate number of cases therapy must be protracted.

The histopathology of these two types of warts is discussed.

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NON-CALCULOUS CHOLECYSTITIS

JOHN J. KREJCI, M.D.

Gallbladder disease is the commonest organic source of digestive complaints. Eliason and Stevens (1) state that 15 per cent of the people in the United States suffer from biliary disease and that this figure is increased to 30 per cent after 45 years of age. Cholecystitis and cholelithiasis are intimately related clinically, pathologically and therapeutically; cholecystitis may be metabolic or infectious, may originate independently of gallstones, may precede the formation of gallstones or may develop consequent to the presence of calculus.

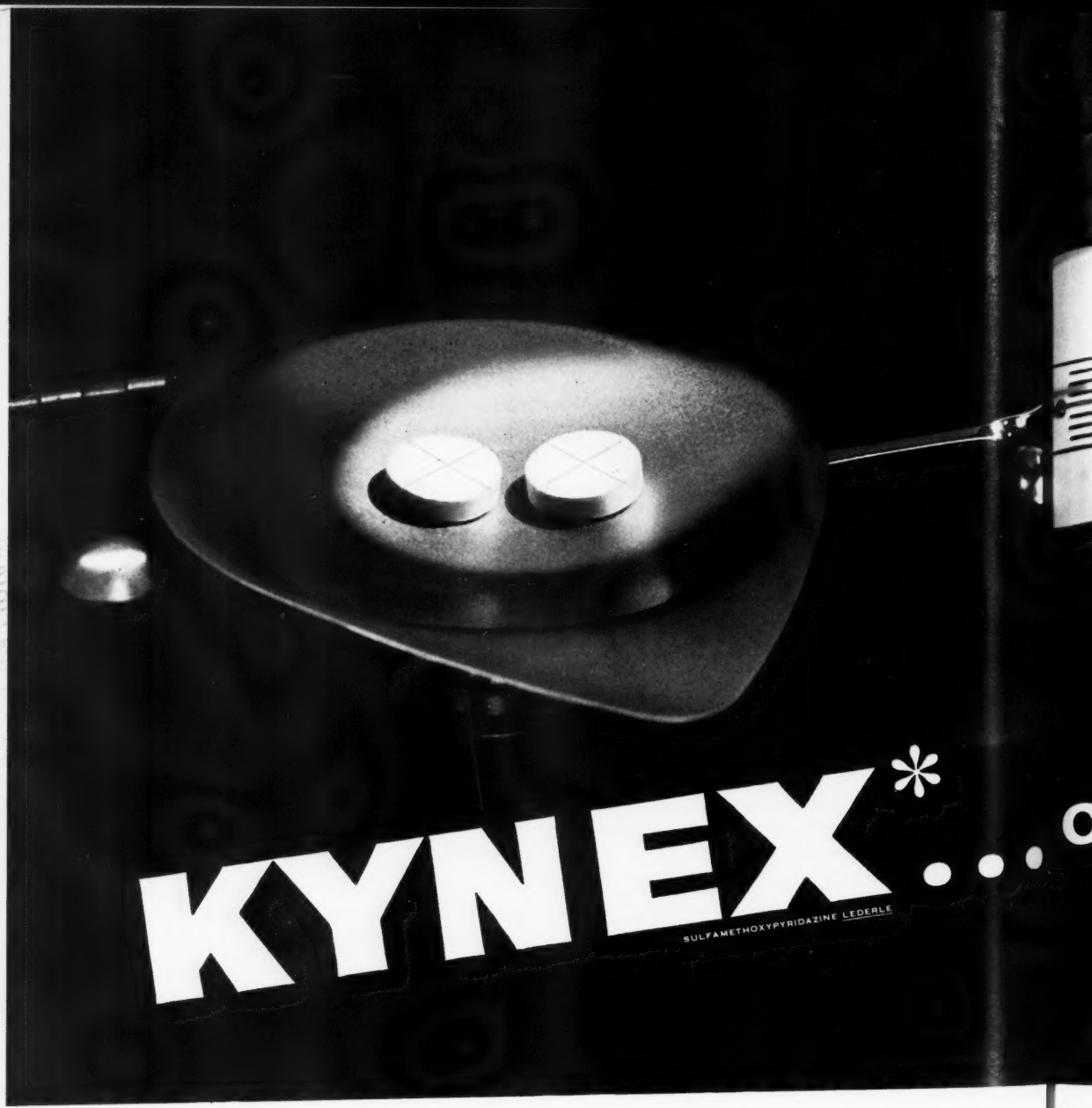
Most recent experimental evidence (2) would indicate that cholecystitis in the majority of instances originates from metabolic derangement and not from inflammation or infection. Disturbance of the absorptive properties of the gallbladder and/or bile stasis results in slow crystallization of cholesterol and/or bile pigments and eventual calculus formation. At any stage in the progression of this disease process, secondary infection may supervene.

Less commonly, a previously healthy gallbladder may become infected by extension from the duodenum by way of the biliary tree, by contiguity with an adjacent diseased viscus, or by lymphogenous or hematogenous extension.

There is a tendency to regard an abnormal cholecystogram as an almost necessary prerequisite to surgery in spite of the clinical history and physical examination indicating definite gallbladder disease. Because of this reluctance to perform gallbladder surgery in the absence of positive roentgenographic evidence of gallstones or of gallbladder dysfunction, it is the purpose of this discussion, first, to stress the true frequency of non-calculous cholecystitis; secondly, to stress the clinical diagnosis of cholecystitis; thirdly, to re-emphasize some of the limitations of cholecystography; fourthly, to urge the more frequent use of the Meltzer-Lyons' technique of non-surgical biliary drainage; and fifthly, to urge the occasional employment of exploratory laparotomy when gallbladder disease cannot be confirmed and when symptoms are intractable to good medical management.

The reported incidence of cholelithiasis associated with cholecystitis is based upon the reported incidence of cholelithiasis in cholecystectomy and this latter incidence as reported in many series of cholecystectomies (3–5), is over 90 per cent. If one were to place credence in these figures, stoneless, or non-calculous, cholecystitis would occur in less than 10 per cent of cholecystectomies and presumably would indicate that non-calculous disease of the gallbladder is an uncommon occurrence. However, it may be that this is not a true index of the frequency of non-calculous cholecystitis since over 80 per cent of cholecystectomies are performed either for gallstones or for complications of gallstones, and other cholecystectomies are performed in association with pancreatic disease. Of more significance, perhaps, would be a statistical analysis of non-calculous and calculous gallbladder disease found at autopsy in people of all age groups who have died of injury or disease unrelated to gallbladder disease. Crump, as quoted by Eliason and Stevens, states "At the post mortem table, stones were found in 33 per cent and diseased gallbladders in 60 per cent of subjects."

In 1934, Mackey (6–8) investigated 264 operative cases of cholecystitis without stones in an attempt to determine those factors which would aid in prognosis; failure to correlate the clinical, radiological and pathological findings led to the impression that "cholecystitis without stones seems to belong to a region on the borderline between functional and organic disease." This attitude illustrates the problem of the patient with symptoms of cholecystitis in the presence of a negative cholecystogram.



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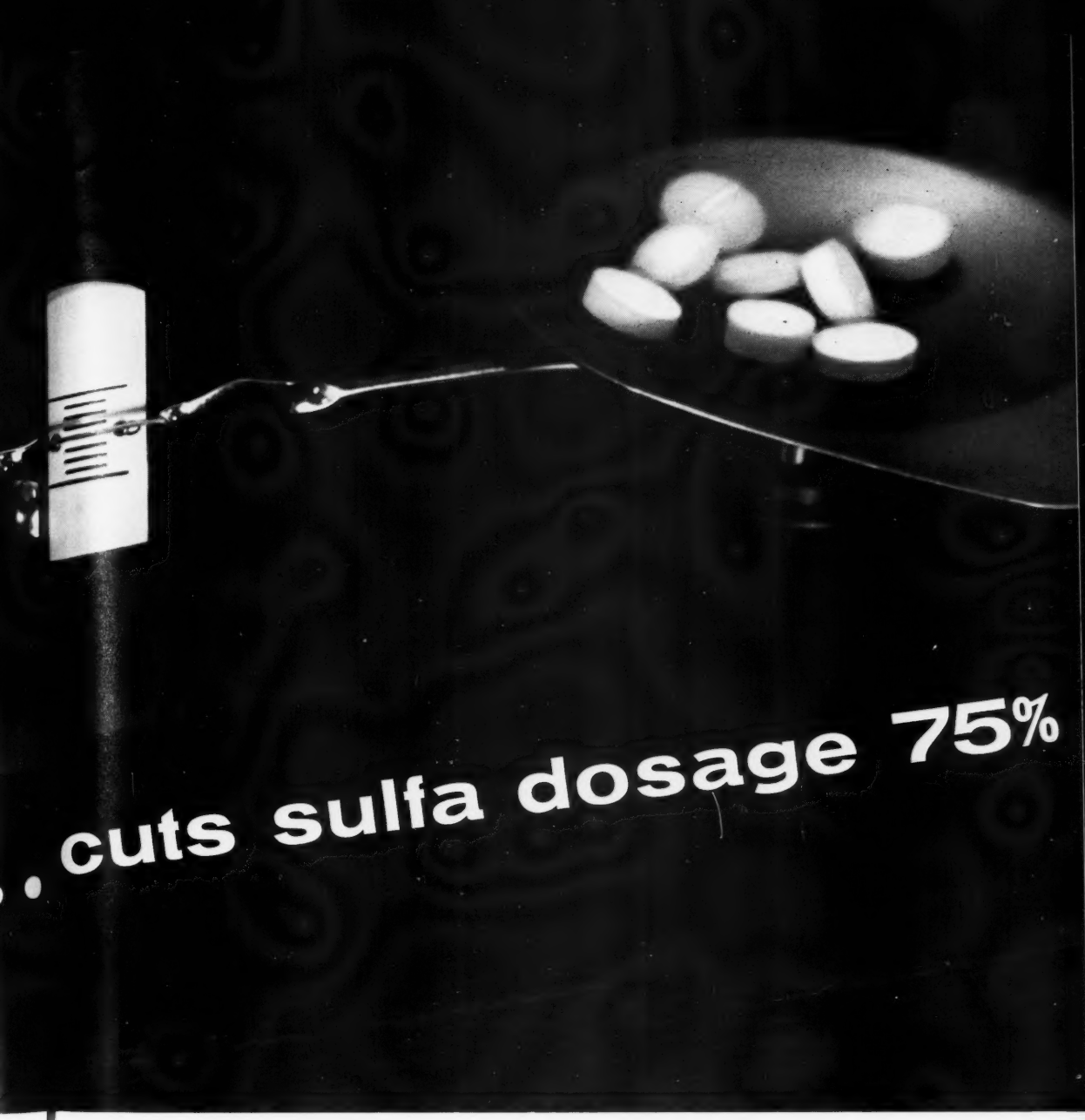

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1. Boger, W. P.; Strickland, C. S. and Gylfe, J. M.: Antibiot. Med. & Clin. Ther. 3:378 (Nov.) 1956.

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The value of a thorough, pains-taking history in the diagnosis of gallbladder disease cannot be over-stressed. The symptom complex in many instances is diagnostic, although certain symptoms are more commonly found in the presence of gallstones.

Today it is generally agreed that the concept of "fair, fat and forty" must be discarded and that the symptoms of gallbladder disease, especially in women, commonly begin in the 18–35 year age group but receive the benefit of definitive surgery as long as 10–15 years after the onset of symptoms. Griffin, at the Mayo clinic, investigated 245 permanent residents of Rochester, Minnesota who had proven gallbladder disease. Fourteen per cent of patients were less than 30 years of age and 28.4 per cent (almost one-third of the total series) were under age 40 (9). Ninety per cent of women with gallstones have borne children and the occurrence of cholecystitic symptoms for the first time shortly after parturition in thin, young women is not uncommon. Attempts to definitely correlate the hypercholesterolemia of pregnancy with the development of simple cholesterosis of the gallbladder or of single or multiple radiate cholesterol stones have not been decisive (10–13).

The predominant complaint in cholecystitis is the symptom complex of indigestion, including flatulence, belching or the inability to belch, eructations, heartburn, epigastric bloating and a sensation of distention after meals. The symptoms are often precipitated by a large meal (14) and by specific food intolerances. However, in addition to dietary indiscretion, prolonged fasting, dehydration, emotional disturbances, overexertion, trauma, a recent operation (15–17) or delivery, change of environment, posture or an unrelated illness (18) (typhoid, septicemia, and following furunculosis) will often be found to be the precipitating factor. In those individuals harboring gallstones, the factor of posture is particularly important to elicit; the occurrence of epigastric or right upper quadrant pain awakening them from sleep and not relieved by the ingestion of bland food or antacids is most significant. Change in environment without change of diet has been important enough to cause the occasional patient to describe his disease as "conventionitis." The occurrence of unexplained fever may be significant in the diagnosis of gallbladder disease, in that this may represent bouts of acute cholangiitis with minimal physical signs.

Cardiac symptoms, such as dyspnea, precordial pain and palpitation often occur during an attack of cholecystitis. The relationship experimentally and clinically, between traction, irritation and distention of the gallbladder and biliary ducts and coronary arterial spasm is well documented and an extensive medical literature testifies to the relief of symptoms of coronary artery disease by performance of cholecystectomy in patients with co-existent disease of the gallbladder (19–26).

Simple cholecystitis may occur with or without pain. Pain, if present, may be epigastric or right subcostal, may radiate around both costal margins and may radiate to the region of the inferior angle of the right scapula or to both scapular regions. This posterior radiation of pain is highly indicative of biliary colic and radiation to the right shoulder often accompanies the pain of acute cholecystitis. Lewis and Peterson (27), in a review of 25 patients with simple cholesterosis, point out that 19 of these 25 patients had pain which was indistinguishable from the pain which occurs with gallstone colic. Five of these patients had normal gallbladder series.

Nausea and vomiting commonly occur with attacks of gallbladder disease. Jaundice may occur with or without gallstones; however, although mild jaundice may occur with acute cholecystitis in the absence of gallstones, deeper jaundice is most commonly caused by choledochal stones.

In general, the physical examination is the least helpful procedure in the diagnosis of chronic gallbladder disease. Following an acute exacerbation of chronic cholecystitis, there is often residual right subcostal tenderness and residual

tenderness to percussion in the region of the inferior angle of the right scapula. Palpation may reveal the enlarged liver of secondary biliary cirrhosis or the enlarged tender liver of acute hepatitis or acute cholangiitis. The gallbladder may be palpable due to obstruction of the cystic or common bile ducts or of the ampullary region.

Although an extensive, accurate history may be sufficient to warrant the diagnosis of cholecystitis, x-ray is a valuable adjuvant. A preliminary scout film of the abdomen may indicate the thick-walled distended gallbladder of acute cholecystitis, the presence of radiopaque calculi, calcareous change in the gallbladder wall, or the gas-filled organ, with or without fluid levels, of acute pneumocholecystitis or pyopneumocholecystitis (28). Cholecystography may be interpreted in several ways: 1) The gallbladder may be normal in size, shape, position, filling and contractability; 2) The gallbladder may fail to visualize; 3) The gallbladder may visualize faintly (one must guard against mistaking the gastric antrum for a faintly filled gallbladder) (29); 4) The gallbladder may be well outlined and suggest the presence of stones; 5) The gallbladder may be well outlined and contain the negative shadows of non-radiable calculi or the negative stellate shadows of gas-filled gallstones (30); 6) The gallbladder may be irregular in outline due to the presence of adenomatous polyps, papillomata or to carcinoma.

It must be remembered that an inadequate dose of dye, or the occurrence of diarrhea or vomiting in response to ingestion of the dye may result in non-visualization or faint visualization of the gallbladder. Hepatic cirrhosis may cause these same unsatisfactory results.

If gallbladder visualization is poor, if the patient had no difficulties due to ingestion of dye and if marked hepatic disease is absent, the oral cholecystogram should be repeated. Increasing the dose of dye may result in visualization. If the gallbladder series is normal, reducing the dose of dye by one-half may demonstrate small calculi which were obscured by the intense concentration of dye.

The position of the patient during cholecystography is important. Erect films and films taken with the patient in the right lateral decubitus position may demonstrate layering of cholesterol stones, which tend to float.

Intravenous cholecystography should be reserved for those cases in whom repeated non-visualization of the gallbladder with oral cholecystography does not satisfy the physician that gallbladder disease is present. It is the procedure of choice in the investigation of the biliary tree following cholecystectomy.

It should be stressed that faulty gallbladder visualization does not necessarily indicate gallbladder disease and that normal cholecystography does not necessarily exclude significant gallbladder disease.

Often, in the absence of all deterring factors, the cholecystogram is inconclusive and the patient must either be investigated further or must be resigned to continuing disability. The next logical investigative procedure is the use of the Meltzer-Lyons' procedure of nonsurgical duodenal drainage. The test, if properly performed, is well tolerated by the patient. Microscopic examination of the bile obtained may show the characteristic epithelium or leukocytes, cholesterol crystals, amorphous bilirubin or the yellowish-brown crystals of calcium bilirubinate. Failure to submit a fresh specimen of bile may result in damage or destruction of its cellular elements, and chilling or prolonged standing may cause precipitation of crystals. A negative biliary drainage is of no value, since neither cholecystitis nor cholelithiasis are necessarily accompanied by crystals in bile. It may be necessary to repeat duodenal drainage on several occasions.

At laparotomy the surgeon must be familiar with the early chronic inflammatory changes present in the gallbladder (31). The gallbladder may lose its typical blue-green transparency, and demonstrate slight opacity and pallor. An excess

of subserous fat may impart to the wall of the gallbladder a white or yellowish opacity. At times, the cystic lymphatic gland is enlarged. There may be a definite subcapsular fibrotic thickening of the liver adjacent to the gallbladder. Many times, there are adhesions of the gallbladder to the pylorus, the first portion of the duodenum, the hepatic flexure and very often to the great omentum. Obliteration of the foramen of Winslow by these adhesions is common. Some of the above pathologic changes are indeed minimal and it is not suggested that cholecystectomy be performed because of the presence of excessive subserous fat or slight enlargement of the cystic lymphatic gland. However, even these minimal changes may assume some degree of importance when evaluated in conjunction with other more definite pathologic changes. The presence of an extrahepatic gallbladder with a pronounced mesentery may indicate the reason for the patient's symptom complex in that this type of gallbladder is particularly prone to torsion. Aspiration of the bile may disclose changes in color, consistency and viscosity which indicate pathologic gallbladder function. At times, the chronic inflammatory changes may be so minimal and yet the clinical history of the patient so indicative of gallbladder disease that one must resort to cholecystotomy, and examination of the mucosa of the gallbladder may demonstrate the heavily rugaed, dull-red mucous membrane and the stippling or "tiger-striping" of cholesterosis, or the presence of polyps, papillomata or carcinoma of the gallbladder.

Cholecystectomy for non-calculous gallbladder disease has been followed by a lesser proportion of cures than has its use in cholecystitis with cholelithiasis. Lewis and Peterson found that, in 20 cholecystectomized patients, 14 were cured, 4 patients were well on a low fat diet and two were not relieved. Mackey reported cure in 14 of 33 patients with cholesterosis without stones who underwent cholecystectomy. In reviewing these and other series of cholecystectomies for non-calculous gallbladder disease (32, 33) one is impressed by the fact that most of these cholecystectomies were performed because of single cholecystograms which reported absent or faint visualization of the gallbladder or "the suggestion" of stones. It is suggested that the utilization of repeated cholecystograms and the more frequent use of duodenal drainage would contribute to better preoperative diagnosis with the performance of fewer poorly-indicated cholecystectomies and an increase in the percentage of post operative cures.

Illustrative Patient Reports

Case Report No. 1. A 40 year old married, nulliparous white female had a history of a right nephropexy for right upper quadrant abdominal pain three years prior to her current admission on September 10, 1955. Post-nephropexy, the character of her right upper quadrant abdominal pain changed; attacks of pain became more frequent and more severe and the patient required frequent, large doses of demerol for control of pain.

Oral cholecystogram was negative.

Biliary drainage: "A" specimen—bile stained epithelial cells, mucus, some cholesterin crystals; "B" specimen—some cholesterol and calcium bilirubinate crystals; "C" specimen—numerous cholesterin crystals, mucus, few epithelial cells.

At operation the gallbladder showed the typical changes of cholesterosis. Cholecystectomy was performed.

Pathology report: The gallbladder exhibits evidence of mucosal hyperplasia. Findings are compatible with adenoma of the gallbladder or chronic cholecystitis glandularis proliferans. There is no evidence of cholesterosis of the gallbladder wall.

Comment

The patient illustrates the problem of a negative cholecystogram and positive duodenal drainage. She has had no significant abdominal pain in the year and one half following surgery.

Case Report No. 2. A 37 year old married, white female was hospitalized on October 21, 1955 with the complaints of frequent, often

nocturnal, attacks of upper abdominal pain, indigestion, acid eructations and occasional nausea and vomiting. All symptoms were of eight years' duration and occurred within one year following delivery of her only child.

Oral cholecystogram was negative.

Duodenal drainage: "A" specimen—many cholesterin and calcium bilirubinate crystals, some epithelial cells; "B" specimen—some epithelial cells, mucus, 7–8 white blood cells; "C" specimen—many cholesterin and calcium bilirubinate crystals, many epithelial cells, mucus, fat cells, bacteria.

At surgery, the gallbladder was thickened, changed in color and peri-cholecystic adhesions completely obliterated the Foramen of Winslow. Cholecystectomy was performed.

Pathology report: There is some edema of the wall with a diffuse lymphocytic infiltration of the muscle layers. The mucosa seems to be eroded at some points.

Comment

This patient also exhibited a normal cholecystogram in the presence of an abnormal duodenal drainage. (The absence of crystals in the "B" specimen was probably due to faulty technique in the performance of the test.) She also illustrates the onset of symptoms of cholecystitis occurring for the first time following delivery.

The patient has been symptom free following cholecystectomy.

Case Report No. 3. A 29 year old white male was hospitalized on November 26, 1955 with the complaints of colicky right upper quadrant abdominal pain referred to the right flank and right inferior scapular region, associated with heartburn and nausea. There was a history of intolerance to fried and fatty foods and to coarse vegetables. All complaints were of five years' duration.

On admission, the rectal temperature was 100 degrees F., leukocytes 18,450, polymorphonuclear leukocytes 88 per cent.

Oral cholecystogram was negative.

Duodenal drainage: "A" specimen—occasional cholesterin crystals; "B" specimen—few cholesterin crystals; "C" specimen—some cholesterin and calcium bilirubinate crystals, epithelial cells.

At surgery, the gallbladder showed minimal thickening and color change. Cholecystectomy was performed.

The pathologist reported a normal gallbladder.

Comment

In spite of a normal pathological report, surgery was followed by prompt decrease of the leukocyte count to normal and the patient has been free of symptoms for over a year.

Case Report No. 4. A 70 year old white male sustained a perforated diverticulum of the recto-sigmoid in February, 1951. A three-stage resection of the sigmoid was performed, culminating in closure of a transverse colostomy on May 15, 1951, following which he was discharged from the hospital on May 26, 1951. Approximately one month later, on June 30, the patient was readmitted with severe right upper quadrant abdominal pain radiating to the left upper quadrant and to the right scapular tip. The temperature was normal, but the leukocyte count was 20,100 and there was a palpable tender mass in the right sub-costal region.

Operation revealed acute cholecystitis with gangrene, and the absence of biliary calculi. Post operative course was uneventful.

Comment

This patient illustrates the occurrence of acute stoneless cholecystitis following unrelated surgery.

Case Report No. 5. A 71 year old white male had been ill at home for several days with apparent Grippe. Six hours prior to admission, the patient developed increase malaise, anorexia, nausea and moderate epigastric pain. Rectal temperature was 100.2° F. The abdomen was tense and slightly distended; there was marked epigastric tenderness and a tense, tender mass in the right upper quadrant.

At emergency surgery, there was incomplete

torsion of an extrahepatic gallbladder suspended by a well developed mesentery. The gallbladder was tightly distended, thin walled and trabeculated due to stretching of its vascular supply. It contained a malodorous, milk-white fluid and no calculi. The post operative course was excellent.

Pathology report: The wall of the gallbladder is slightly thickened and the mucosa is infiltrated with round cells.

Comment

This case demonstrated the unusual picture of acute obstruction of the gallbladder due to spontaneous torsion and also the occurrence of cholecystitis during incidental infection.

Short and Paul (34) in a series of 53 cases of gallbladder torsion, found only 6 cases in males. They suggested that a gallbladder palpable with varying facility from time to time may indicate the diagnosis.

Case Report No. 6. A 24 year old divorced white female, gravida I, para 1–0–0–1 was hospitalized on March 16, 1955 with the complaints of intolerance to fried fatty and greasy foods, fullness and bloating, and frequent attacks of colicky right upper quadrant abdominal pain radiating to the right scapular tip.

Oral cholecystogram was negative.

Surgery was performed and the gallbladder was thickened, dull grayish pink in color and contained two small pigmented stones. Cholecystectomy was performed.

Pathology report: Sections show phantom glandular structures. The wall is thickened, fragmented and sparsely infiltrated with chronic inflammatory cells.

Comment

This case, despite the presence of biliary calculi, is included because it illustrates the problem of a patient with onset of symptoms of cholecystitis, including true biliary colic, following upon pregnancy. In spite of a negative cholecystogram, surgery was performed with the indication of a good clinical history.

Case Report No. 7. A 45 year old married, multiparous white female had a three year history suggestive of gallbladder disease. There were almost constant symptoms of abdominal fullness, heartburn, and belching. In spite of good medical treatment, the patient developed frequent attacks of epigastric pain, beginning in the right hypochondrium and radiating across the upper abdomen, around the right costal margin to the right scapular tip and into the right shoulder. Recent nausea and vomiting had accompanied the attacks. The patient claimed to have had "dark urine and watery-like stools" during her attacks; there was no history of jaundice.

Oral cholecystograms on several occasions were negative. A gastro-intestinal series was negative.

At operation, the gallbladder appeared normal. There was a congenital, broad, flat, taut peritoneal fold which angulated the second portion of the duodenum and displaced it to a position beneath the right lobe of the liver. The peritoneal fold was quickly severed and the partial duodenal obstruction released. The patient has been free of symptoms for over five years following surgery.

Comment

Following diagnostic studies of this patient, one was forced to choose between discharge of the patient to continuing disability and the performance of exploratory laparotomy. If the probability of functional gastro-intestinal disease can be excluded, occasionally, but only occasionally, intractability of symptoms will necessitate surgical exploration.

In another such patient, annular pancreas was found to cause partial duodenal obstruction.

Summary

A discussion of non-calculous cholecystitis is presented.

1) The true frequency of stoneless cholecystitis may be higher than generally reported.

2) The basic diagnostic weapon of the physician in the diagnosis of cholecystitis is a searching clinical history.

3) The limitations of cholecystography are stated and its position as a diagnostic aid is emphasized.

4) The more frequent use of non-surgical biliary drainage should be encouraged as a further step in diagnosis in the presence of a negative cholecystogram.

5) The very occasional employment of surgical exploration in the diagnosis of gallbladder disease is advised.

Illustrative case reports are presented.

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CARCINOID

Report of Three Cases

WILLIAM J. SUPIK, M.D.

Coincident with the recent popular interest in Carcinoid tumors, three such cases were discovered at the St. Joseph's Hospital within a period of several weeks, and, in typical fashion, defied diagnosis until examination of the tumor following surgical removal.

From an early concept of a simple benign tumor almost always occurring in the appendix present knowledge now brands Carcinoid as a true malignant neoplasm, able to metastasize, though with apparent reluctance to do so at times, and with the additional characteristic of endocrine-like function which produces a rather unvarying group of symptoms referred to as the Carcinoid Syndrome.

It is now generally accepted that Carcinoid tumors arise from the Kulchitsky cells of the crypts of Lieberkuhn of the gastrointestinal tract. They have been reported as occurring in the stomach, gall bladder, duodenum, small bowel, Meckel's diverticulum, appendix, ileocecal valve, cecum, colon, rectum and in the gastrointestinal components of teratomas of the ovary. The sites of greatest frequency are the appendix and ileum.

The gross appearance of the Carcinoid tumor varies somewhat according to location, but characteristically appears as a small, firm, flat, sharply outlined submucosal nodule covered by intact mucous membrane. The cut surface reveals a smooth homogeneous mass of light grey yellow color, rather rubbery in consistency. The tumor originates in the submucosa and may later extend to and through the muscularis, finally involving the serosa. The mucosa is rarely invaded but is pushed inward to form a polypoid or sessile projection into the lumen of the bowel. An exception to this general trend may be noticed in Carcinoid tumors of the rectum which may ulcerate. One common characteristic with enlargement of the tumor is a marked fibrotic reaction with a tendency toward kinking or knuckling.

Until this stage in the development of the Carcinoid tumor, symptoms, if at all present, will be those symptoms typical of the mechanical effect of any mass interfering with proper bowel function, namely, partial or complete intestinal obstruction, intussusception, and, in the rare instances of ulceration, rectal bleeding. Rectal bleeding may occur with the intussuscepting type of lesion, not from the tumor itself but from the normal mucosa of bowel involved in the intussusception. Only in the rare cases of ulceration does rectal bleeding lead to their discovery. Most frequently, rectal Carcinoid is entirely asymptomatic and is discovered on routine proctosigmoidoscopy. Carcinoid of the stomach, also, is in most instances, asymptomatic and is more often discovered as an incidental finding.

Because of their location in an area or in areas not likely to interfere with normal gastrointestinal functions for quite some time, Carcinoid tumors may produce secondary metastatic deposits which may grow silently to massive size, especially in the liver. Metastases are usually confined to the abdominal cavity. It is after the development of metastases that the Carcinoid becomes a functioning tumor and a new set of clinicopathological features is added to the mechanical effects of the tumor itself. The amino acid tryptophane, which normally converts about 1 per cent to serotonin, undergoes a change in its utilization and converts as much as 60 per cent to serotonin. This parasitic action upon the tryptophane stores of the patient results in a diminution in the amount of this essential amino acid available for other metabolites such as

protein and niacin. The known action of serotonin, to stimulate smooth muscle, may explain the hypermotility of the intestine with resulting chronic diarrhea, weight loss and other related nutritional disturbances, and the respiratory distress, but fails to account for the occurrence of the peculiar cutaneous flushes and valvular heart disease. The diagnosis of a functioning Carcinoid tumor may be confirmed by testing the urine for the excreted serotonin metabolite, 5-hydroxyindole acetic acid, a positive test being indicated by a purple layer developing on the specimen.

The treatment of Carcinoid, when possible, must always consist of complete extirpation, and will vary from simple local excision to radical removal depending upon size, location and extent of tumor.

Case Number 1

A 58 year old white male was hospitalized on September 5, 1956 with complaints of diarrhea and intermittent abdominal cramps of two weeks' duration. He was in no acute distress and demonstrated a normal pulse rate, a low-grade fever of 99°F., and a leukocytosis of 11,350 with a normal differential. Barium enema revealed an irregular, polypoid filling defect in the region of the cecum, interpreted as a "probable carcinoma of the cecum." Right hemicolectomy, terminal ileectomy and ileo-transverse colostomy was performed. Gross metastases were not demonstrated. Pathological report: Carcinoid of the Cecum. Post-operative convalescence was uneventful.*

Case Number 2

A 68 year old white female was hospitalized on November 7, 1956 with complaints of intermittent, colicky lower abdominal pain and vomiting, of three weeks' duration. She was afebrile, with normal hematologic studies; urinalysis was negative. Pertinent physical findings consisted of tenderness of the lower abdomen chiefly centered about a scar which was the result of hysterectomy performed twenty years ago, and the presence of hyperactive intestinal sounds. There were no roentgenographic studies performed. The tentative diagnosis was partial intestinal obstruction due to intra-abdominal adhesions and surgery was performed on the day following admission. Abdominal exploration disclosed a severely constricted segment of ileum approximately 20 cms. from the ileocecal valve and a group of pathological lymph nodes in the mesentery of the involved loop. These were resected with the tumor mass and continuity of the ileum reestablished. The pathological report revealed the tumor to be an "undifferentiated Carcinoid of the ileum." The patient was discharged following an uneventful recovery.

Case Number 3

A 44 year old white female was hospitalized on November 6, 1956 with complaints of intermittent abdominal cramps and rectal bleeding of two years' duration. In the early course of the disease, the episodes of pain and bleeding were mild and very infrequent, with long asymptomatic intervals; however, more recently, symptoms occurred with greater frequency and intensity and bleeding became more profuse. Between April 1956 and hospitalization in November 1956, proctosigmoidoscopies, gastrointestinal series, barium enemata and air contrast studies were performed repeatedly with consistently negative results. However, the most recent proctosigmoidoscopic examination, although revealing no intrinsic lesion, did report the presence of dark, clotted blood descending from the higher bowel. Operation was performed eleven days after admission and revealed intussusception of the terminal ileum containing a tumor, into the cecum. A solitary metastatic nodule 2.5 cms. in diameter was densely adherent to the mesenteric border of the ileum. The remainder of the abdominal exploration was negative. Right hemicolectomy, terminal ileectomy and ileo-transverse colostomy was performed. Pathological report described Carcinoid of the

* With regard to Case #1, the serotonin test was negative on January 21, 1957.

ileum with regional metastasis in the mesenteric lymph node. Microscopically, there was also diffuse scarring and chronic inflammation of the mucosa of the cecum. After an uneventful recovery the patient was discharged; urinary tests for functioning Carcinoid tumor was performed on January 17, 1957 and was negative.

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OFFICE BRONCHOGRAPHY

BERNARD S. KLEIMAN, M.D.

Bronchography is a procedure which frequently necessitates an overnight hospital stay. The purpose of this article is to outline a method which can be performed as an office procedure. It has proved to be basically safe and rapid and provides sufficient bronchographic detail to allow the physician to evaluate the possibility of bronchiectasis or of bronchial obstruction. It is intended primarily as a good "scout" procedure and not as a substitute for the detailed bronchography which is a necessary prerequisite to good thoracic surgery. Since laryngeal visualization is not necessary, this procedure can be carried out successfully without prior detailed technical training.

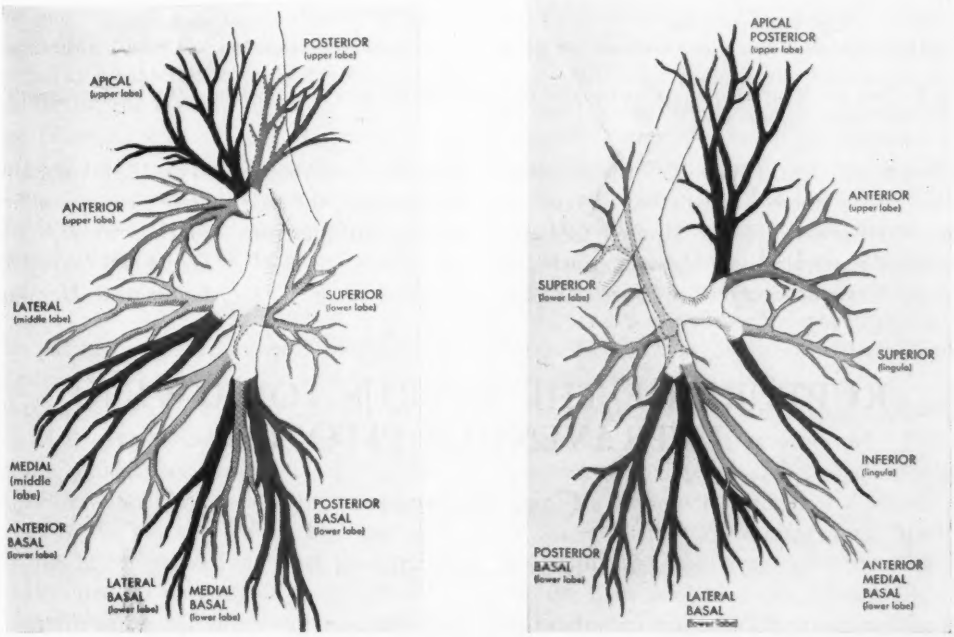
PROCEDURE

Anesthesia:—Preoperative medication consists of Nembutal 0.1 Gm. and Hycodan bitartrate 5 mgm. by mouth one hour prior to the procedure. Atropine Sulphate 0.4 mgm. is administered hypodermically one-half hour prior to starting. At the time of bronchography 2 per cent Pontocaine is sprayed on the pharynx, hypopharynx and nasopharynx. (It is anaesthesia of the nasopharynx which appears to have the greatest value in controlling the gag reflex.) This pharyngeal spraying is repeated at one to two minute intervals until the gag reflex is lost. Neo-synephrine 1 per cent followed by Pontocaine 2 per cent is sprayed intranasally along the floor of the nose. If there is an obvious septal deviation, it is better to use the larger nasal chamber. Cocaine hydrochloride (4%) 4–5 cc. is next nebulized, using oxygen as the propelling force, through a DeVilbiss #640 atomizer. Inhalation by mouth carries deeply into the lungs. The limit of 5 cc. of cocaine medication is never exceeded; 2.5–4 cc. usually suffices. (I would caution against the use of Adrenalin or Protocaine in the tracheo-bronchial tree; severe reactions have attended the use of these drugs. Although no anesthetic agent can be used without some degree of risk, cocaine has been found

to be least toxic.) Intravenous barbiturates and oxygen should always be available. Oxygen will quickly relieve nausea and apprehension. The intravenous barbiturates should be used only for impending convulsions; its use has not been found necessary in the past two years. A soft rubber catheter (approximately 16 French) is threaded through the anesthetized nasal chamber until it emerges in the oropharynx. The patient is then asked to sit erect, slightly forward and to grasp the tongue. The catheter is passed blindly into the larynx. This is a surprisingly simple procedure; it is only necessary to keep the catheter in the midline of the oropharynx to prevent it from lateral deviation into the pyriform sinus.

The patient is then placed on the X-ray table. He is rotated to an oblique position with the right chest inferiorly and the left posterior chest wall facing superiorly. Oily Dianosol 15 cc. is instilled through the nasal catheter. This product may be used at room temperature; heating and chilling are not necessary. After three minutes the patient is asked to stand against a chest film holder in a left anterior oblique position, i.e. the left chest wall is positioned against the casette and the right posterior chest wall faces the X-ray tube. A film is taken and the patient is repositioned on the table in approximately the same position in which he last faced the casette, i.e. obliquely, with the left posterior chest wall inferiorly and the right posterior chest wall facing superiorly. Once again Dianosol 15 cc. is instilled; as on the right side, there is a three-minute wait following which the patient is again positioned upright before the casette and an erect right anterior oblique film is taken.

These two films, right and left anterior oblique,



A

Left anterior oblique
(showing right lung)

B

Left anterior oblique
(showing left lung)

FIG. 1. These two illustrations are by Dr. J. Stauffer Lehman and Dr. J. Antrim Crellin as published in Medical Radiography & Photography, Vol. 31, No. 2, pp. 81–83, 1955.

A
Right anterior oblique
(showing the left lung in a case of situs inversus)

B
Left anterior oblique
(showing the right lung, and basular bronchiectasis, in a case of situs inversus)

FIG. 2. These two X-ray illustrations are from the case report by Dr. John Krejci and the author on Kartagener's Syndrome in this issue of this journal.

usually are sufficient. If possible, it is of advantage to review them immediately in order to decide whether more oil is required to better outline any particular lung segment or whether postero-anterior or lateral X-rays are desirable. However, as demonstrated on the accompanying illustrations, the two oblique views are sufficient to delineate the entire bronchial trees.

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RUPTURE OF THE UTERUS FOLLOWING INTRAVENOUS PITOCIN

Case Report

JOSEPH A. KNELL, JR., M.D.

All modern textbooks of obstetrics warn of the danger of ruptured uterus due to the use of Posterior Pituitary Extract (1, 2, 3). There are numerous case reports of the occurrence of this accident in the obstetrical literature. Mendenhall reviewed the world literature up to 1929 and collected 89 cases of rupture of the uterus due to pituitary extract (4).

In 1928, Kamm and his co-workers in the laboratories of Parke, Davis and Company,

separated Posterior Pituitary Extract into the pressor and diuretic fraction known as Pitressin, and the oxytocic fraction known as Pitocin. Since the introduction of the purified and standardized Pitocin, there have been few reported cases of rupture of the uterus due to its use. Eastman, commenting on a paper by Burnett in 1951 said, ". . . . with the increasing use of intravenous pitocin I can see right now the writing on the wall, not only on the wall but all over the ceiling and floor. In my sleep I can see it in the form of a paper that is certain to appear one of these days with some such title as: 'A Case of Rupture of the Uterus Following Intravenous Pitocin'" (5). The first such case was reported by Schneider, Ferguson and Miller, in a paper describing their experience with the use of Pitocin (6). A second case of rupture of the uterus due to Pitocin was also reported by the same authors (7). Gready mentioned a case of ruptured uterus occurring in a grand multipara following the use of Pitocin, but this patient was also subjected to an internal version which probably accounted for the rupture (8).

The following case is thus the third to be reported of rupture of the uterus due to Pitocin. This case is presented because of its unusual nature and also to once again bring out the danger of using Pitocin in the grand multipara.

Case Report

The patient was a 42-year-old, white housewife, para 9-0-1-9, who was first seen in the prenatal clinic on March 23, 1955. She stated that her L.M.P. was sometime in October, 1954. However, quickening was noted in January, 1955, and examination revealed the uterus to be enlarged to the size of a 28–30 week gestation. Therefore her estimated date of confinement was calculated to be early in June, 1955. On the initial visit her blood pressure was 208/110, there was 4+ ankle edema but no albuminuria. Her weight was 267½ pounds which was about 17 pounds more than her pre-pregnant weight. The remainder of the physical examination was normal. Her past obstetrical history was indefinite. She stated that her babies weighed between 6 and 10 pounds at birth, that they had all been full term, and that she had had easy labors with spontaneous deliveries. She thought that she may have had hypertension with her seventh pregnancy in 1948, but this was not confirmed.

The patient was admitted to the hospital the following day for complete evaluation. Her laboratory studies on this admission were as follows: Hgb. 83 per cent; R.B.C. 4,320,000; leukocytes 9,700. Urinalysis; acid reaction; 1.010 specific gravity; negative albumin, negative sugar, 5–8 W.B.C., 3–5 epithelial cells, and 1 granular cast. Blood sugar was 95 mg. per cent, N.P.N. 48; Uric acid 4.5. A P.S.P. test showed, first specimen 54.6 per cent excretion, and second specimen 7.5 per cent excretion. A Fishberg Concentration Test showed first specimen 1.024, second specimen, not enough urine, and third specimen 1.020. A chest x-ray was reported as showing a normal size heart and aorta, and clear lung fields. Ophthalmoscopic examination revealed normal eyegrounds. A diagnosis of chronic essential hypertension without cardiac or renal damage was made. She was treated with bed rest, salt-free diet, Diamox and Serpasil. She responded to therapy and within six days her B.P. had stabilized at 150/100, her ankle edema had subsided and she lost 12 pounds in weight. She was discharged on March 29, 1955, on the above therapy, to be followed in the pre-natal clinic.

She was seen weekly until April 25, 1955, at which time her blood pressure was 210/130, there was 4+ albumin and 2+ ankle edema. Through a misunderstanding, the patient was not admitted to the hospital until April 29, 1955. On this date her blood pressure was 170/100, there was 2+ ankle edema, 4+ albumin and she weighed 265 pounds. She was complaining of severe, constant frontal headaches and epigastric pain. Examination revealed that the vertex was floating and the estimated size of the fetus was 5 pounds. The fetal heart

was not heard. The cervix was soft, but uneffaced and not dilated. The patient was treated with bed rest, salt-free diet, diuretics, sedation with Magnesium Sulfate and Serpasil. Under this therapy her blood pressure varied between 140–200/100–120, her albumin remained at 4+, her edema subsided, her headache subsided and her urinary output remained good for the next 24 hours. The fetal heart could never be heard during this time and x-ray of the abdomen on May 2, 1955 confirmed the impression of fetal death. Because of this patient's severe pre-eclampsia, superimposed on chronic hypertensive vascular disease it was decided that she should be delivered. Therefore a medical induction with Quinine sulphate, Castor Oil and enemata was begun at 12:30 P.M. on May 2, 1955. Mild irregular contractions promptly ensued. At 1:30 A.M. on May 3, 1955 the cervix was 50 per cent effaced and 2–3 cms. dilated with the presenting vertex engaged. An amniotomy was then performed. By 9:30 A.M. of the same day, her contractions had ceased and there was no change in the condition of the cervix. Her B.P. was 170/100 and urine showed 4+ albumin. Although we were fully cognizant of the danger of using Pitocin in the grand multipara, nevertheless we felt that the condition of the patient justified the risk of using it.

At 9:30 A.M., 5 Minims of Pitocin in 500 cc's. of 5 per cent Glucose in distilled water was started intravenously at the rate of 16 drops per minute. At 11:30 A.M. the patient was having good regular contractions lasting 45 seconds and recurring every 2–3 minutes. The cervix was 4 cms. dilated, 75 per cent effaced and the presenting part 2 cms. above the spines. The Pitocin infusion was then slowed to 8 drops per minute. Suddenly at 12:20 P.M. her labor became tumultuous and she spontaneously delivered a stillborn female child.

The Placenta was expressed with difficulty. Bleeding was minimal and the patient did not complain of abdominal pain. She was taken immediately to the delivery room. Examination revealed there was a laceration of the left side of the cervix which extended completely up through the lower uterine segment. The examining hand could be introduced through this rupture into the left broad ligament. The remainder of the uterus was found to be intact. The condition of the patient at this time was excellent. She was not in shock, bleeding was minimal and her blood pressure was 170/100. Whole blood infusion was immediately started and the patient was taken to the operating room where a total hysterectomy and bilateral salpingo-oophorectomy was performed. The patient withstood the procedure very well. She had an uneventful post-operative course. Her blood pressure gradually stabilized at 140–160/90–100, but her urine continued to show a 4+ albumin, even on discharge on her ninth post-operative day. She was instructed to return to the clinic for follow-up, but failed to return.

Comment

Contrary to experience, this patient's pre-eclampsia did not improve with the intrauterine death of the fetus. Therefore we felt justified in using Pitocin to induce labor. We anticipated the uterine rupture and therefore two pints of blood had previously been typed and cross-matched and was in the delivery room, and the Pitocin infusion was started through an 18 gauge needle. The uterus was explored immediately after delivery and thus there was no delay in making the diagnosis or instituting therapy. In retrospect, however, we feel that if the Pitocin were discontinued when labor was well established at 11:30 A.M. on May 3, 1955, rupture of the uterus might not have occurred.

Summary

A review of the literature discloses only two previously reported cases of rupture of the uterus due to the use of Pitocin, although there are many references to this accident occurring with the use of Posterior Pituitary Extract.

Another case of rupture of the uterus due to the use of Pitocin is presented.

Pitocin should be used with extreme caution, if at all, in the multipara who has had four or more full term pregnancies.

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SURGICAL TREATMENT OF PULMONARY EMPHYSEMA

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Whether or not there is enough measurable scientific data to support what is herein reported is open to discussion. Increased pulmonary ventilation as shown by increased breath sounds, increased motion of the chest and diaphragm, and increased inability to work and to carry on other activity provide ample clinical evidence to prove the beneficial effects that surgery can achieve for the emphysematous patient whose condition is no longer satisfactorily controlled by medical treatment.

Pulmonary emphysema is not a concise, easily defined disease entity. Clinically and pathologically there are many variables. At one end of the gamut of this disease there is bronchial asthma and at the other end there is localized cystic disease of the lung.[1] Pulmonary emphysema is found somewhere in between these two extremes and is associated with a greater or lesser degree of cor pulmonale.

To each worker in this field pulmonary emphysema doubtless has a rather different meaning. To the author a patient with pulmonary emphysema can be described as follows.[2] The patient is usually thin, since it requires considerable effort to breathe and the shortness of breath makes it difficult to eat properly. He coughs upon awakening in the morning. He expectorates a small amount of tenacious sputum, usually requiring from one to three hours to clear it away. When the bronchial secretions are removed by cough, the patient is relieved of further cough but he is exhausted. He becomes dyspneic on slight exertion. The amount of exertion possible depends upon the degree of emphysema. He often purses his lips on expiration and leans on his elbows in order to breathe easier. If an upper respiratory infection occurs there is a greater degree of dyspnea.

Upon examination the chest is found to be in a state of expansion or is barrel-shaped. The motion of the chest is limited but equal. Breath sounds are greatly diminished and there is rarely a bronchospastic element. Immediately after cough the breath sounds are further diminished or disappear completely. This finding is characteristic of pulmonary emphysema. The diaphragm moves only slightly upon percussion.

A chest roentgenogram discloses few lung markings. The diaphragm is low and often flattened, and may show a step-like contour. The ribs are rather far apart, revealing wide intercostal spaces. Fluoroscopy shows reduced motion of the diaphragms and there may be a shift of the mediastinum on inspiration and expiration, thus revealing greater obstruction to air flow in one lung as compared to the other.

In a pathologic study[3] there is noted thinning out of the alveolar walls and septa. Alveolar dilatation is revealed and often there is an alveolar breakdown resulting in coalescence of the alveolar spaces. Coalescence of sufficient broken down alveolar spaces causes cystic-like blebs or bullae. The elastic fibers of the lung disappear and break into pieces. The lung becomes voluminous and thus inelastic, holding the chest in a state of expansion and the diaphragm lower than normal. Even in so-called generalized pulmonary emphysema the pathologic changes are not uniform. All areas of the lung are not equally involved by the pathologic process, yet there is rarely any area of lung that can be considered normal. The areas of lung showing the greatest pathologic destruction are the most functionally useless areas. Nevertheless, some areas are more nearly normal than others. As the alveolar air sacs become dilated and larger, air cannot leave them as easily. There is increased air pressure in the coalesced, broken down alveolar spaces. This causes compression of the more nearly normal surrounding areas of the lung. The voluminous lung, occupying a larger than normal space, and the loss of elastic tissue permit collapse of the bronchi. Normally the bronchi remain open by negative intrapleural pressure that holds expanded the elastic lung, the elastic fibers of which exert a circumferential pulling force upon the bronchi. Thus, the free flow of air is impaired by the tendency of the bronchi to collapse, particularly on expiration. Ordinarily this collapse on expiration is a carefully timed procedure and when it occurs slightly quicker than normal it tends to trap air in the alveoli. This factor is found to be greatest in the most diseased areas, therefore, tidal air is not mixed uniformly.[4] Bronchial spasm, the presence of secretions, and edema or infection of the bronchial wall will further impede or obstruct the flow of air through the bronchi. The result is impaired ventilation. Tidal air volume is reduced. Residual air and functional residual air are increased.

The capillaries of the alveolar walls undergo degenerative changes and disappear with the breakdown of alveolar spaces. There is reduced blood flow and increased pulmonary artery pressure. The prolonged increased pulmonary artery pressure gives rise to thickening of the intima, additional reduction of blood flow, and further increase of pulmonary arterial pressure. Thus, a vicious cycle is initiated.[5] The final result is cardiac failure with cor pulmonale.

All phases of the physiology of respiration are altered to a greater or lesser degree. Ventilation is reduced, as indicated by diminished to absent breath sounds and reduced motion of the chest and diaphragm. The passage of gas across the alveolar membranes is impaired, altering the exchange of carbon dioxide and oxygen between blood and alveolar air. Circulation is reduced by degenerative changes in the capillaries, which further interfere with the proper exchange of gases. There is an altered carbon dioxide and oxygen tension in the blood. This will affect the respiratory control centers in the central nervous system. The normal control and regulation of the complex phases of respiration are numerous and complicated. It is not within the scope of this article to discuss the various aspects of respiration, but they are graphically indicated in Fig. 1. The surgery of pulmonary emphysema probably affects many phases of the complex respiratory control mechanisms. However, it primarily affects ventilation of the lungs and produces better and more uniform mixing of the tidal air. It also affects inflation and deflation of the lung.

Patients who have pulmonary emphysema will respond to medical therapy in the early stages of the disease.[6] However, when medical therapy is not effective in relieving dyspnea and cough, and when further progression of the disease is not stopped or retarded, the patient should be given the benefits that surgery offers. Of course, the younger the patient and the better the cardiorespiratory system, the greater will be the results of surgery. Surgery cannot create a new lung, but it can permit a functioning lung to do its work of respiration. Therefore, the more nearly normal the lung, the better will be the surgical results. If the heart shows signs of cor pulmonale and this is not associated with coronary artery disease or hypertension, there is no contraindication to surgery as long as the patient is not in cardiac failure. Perhaps the contraindications to surgery are age above sixty-five, myocardial degenerative disease, especially with hypertension, and certain associated diseases, particularly bronchiectasis. Cor pulmonale is not a contraindication provided the patient is not in failure at the time of operation.

The first stage in the operation is done on the side showing the greater evidence of disease. About three months later it is carried out on the other side. The patient always receives the greatest subjective relief of symptoms from the first operation. The operation is planned to reduce the volume of the lung by removing the least functioning areas of the lung and by surgically denervating the lung.

The reduction of lung volume is accomplished by the clamp and suture method. The lung is actually trimmed away by removing as much of the functionally useless areas as is compatible with the reduced lung size or volume desired. A lobe or segment as such is never removed. Since there will perhaps be no really normal lung tissue present, it is obvious that the entire diseased lung cannot be removed and this should not be attempted. The volume should be reduced to the point where the lung will fit the pleural cavity. The functionally useless lung that must be retained acts as a filler of the pleural space. In view of the fact that the lung has lost its elasticity, great care must be exercised not to reduce the volume too much because it will not expand and fill the pleural space as normal lung tissue does. This fact is real evidence that the elastic tissue has been destroyed. Every caution should be exercised to prevent air leaks from the lung since this may lead to serious postoperative complications.

The reduction of lung volume accomplishes three distinct results: I. It allows expansion and functioning of the more nearly normal areas of the lung since the compressive effects of the useless nonfunctioning areas are at least partially removed. When the compressive effects are eliminated air is permitted to flow more readily through the tracheobronchial system and into the alveoli. It also allows better capillary blood flow. II. Reduction in lung volume restores a more nearly normal negative pressure within the thorax and asserts a circumferential pull upon the bronchi, thus tending to hold them open.

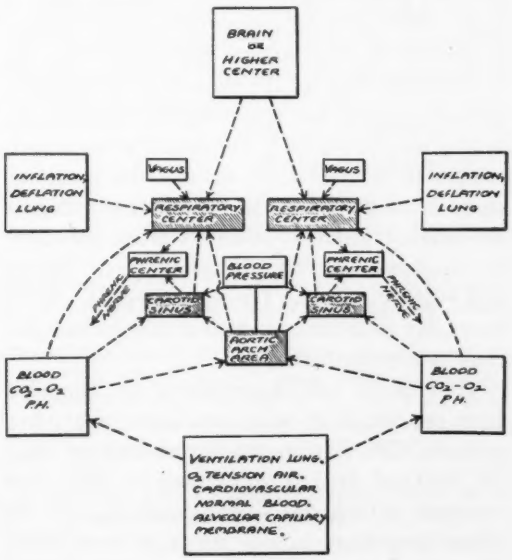

FIG. 1

The restoration of nearly normal negative pressure within the thorax aids venous flow to the right side of the heart. III. Reduction in lung volume allows elevation of the diaphragm and a less expanded state of the thoracic cage, which in turn permits motion of the chest and diaphragm. All of these results effect better and easier passage of air through the tracheobronchial system and thus into and out of the alveoli of the lung. In this way ventilation is improved considerably as evidenced by a return of normal to nearly normal breath sounds, where formerly the breath sounds were absent to nearly absent. It is also interesting to point out that the breath sounds do not disappear immediately after cough as they did before operation. There is also a reduction in residual air, which in turn increases alveolar ventilation.[7]

The denervation procedure[8, 9] consists of removal of the posterior pulmonary plexus by resection and ligature of all branches of the vagus nerve to the lung, heart, and mediastinum. Only the fibers to the esophagus and lower intestinal tract are spared. A sympathectomy of the periarterial, perivenous, and peribronchial type is done. The bronchial artery is resected in order to remove the sympathetic fibers that it carries to the lung. Neither the thoracic or other sympathetic ganglia nor the sympathetic trunk are in any way disturbed.[10] The denervation accomplishes three results: It reduces the tenacious secretion of the bronchial glands, thus reducing the cough. It reduces pulmonary artery pressure[11] by capillary dilatation, increasing the circulation and slightly relieving the cor pulmonale. It relieves any bronchial spasm that may be present, thus aiding ventilation.

These data and observations are compiled from the results of operations upon twenty-five patients. The first operation was done on May 18, 1950 and the last on October 21, 1956. The youngest patient was sixteen years old and the oldest sixty-four years of age; four were above sixty years of age and five were under thirty-nine, the remaining patients being in the forty to fifty-nine year age group. All except two of the patients were male. There were five deaths in the hospital following operation. Among those who survived operation all have been helped except one who died about four months postoperatively without being benefited by surgery. Before operation every patient had for a long time been unable to work. After operation fourteen returned to some kind of work. Those who have not returned to work show a definite increase in exercise tolerance.

The operative deaths resulted in the following manner. Two died on the hottest day in the summer of 1953, one within an hour of operation and the other on his seventh postoperative day. Two deaths were caused by insufficient functioning lung tissue for support. Perhaps if a simultaneous bilateral operation could have been done, they would have survived. One of these two is included in the two who died on the hottest day in 1953. Two patients died because too much lung volume was removed, a mistake in judgment when it was not fully realized that the over-distended inelastic lung would not further expand to fill the pleural space. It was impossible to control the air leaks since the pleural space could not be obliterated. One patient died of acute suppurative bronchitis, but he was also one of the two in whom there was too much reduction of lung volume.

It is difficult to predict before operation who will receive the best results. Unfortunately, pulmonary function studies have not been made on many of the patients. However, it is obvious that pulmonary function studies cannot measure the potential lung function that may be restored. In some patients, even though preoperative studies revealed a generalized disease there was found a nearly normal lobe in a collapsed state caused by the compression of an enormously enlarged companion lobe, Such patients receive considerable relief of symptoms. Thus, one can report that the more localized the pathologic process in the

lung, the better will be the results of surgery. Laminographic studies of the lungs will furnish evidence of the good lung tissue and the destroyed lung tissue. Bronchoscopy and bronchography are of great assistance in eliminating patients with associated disease and should always be used if there is any question as to the presence of other disease, especially bronchiectasis. If associated disease is present and localized, it is in no way a contraindication to surgery since the removal of the pathologic process can be carried out as a part of the reduction in lung volume. It corresponds to the trigger area of Abbott.[12]

The patient with other pulmonary disease and pulmonary emphysema presents a difficult problem. The prognosis of surgical treatment becomes more difficult. The surgical risk is usually increased. If the associated disease does not occupy too great a volume its excision can be part of the reduction in lung volume and does not add to the risk. If the associated disease is one that has contracted and destroyed a lobe and thus reduced its volume, while the remaining lobe has become emphysematous, it seems certain that surgery can offer no aid to the patient.

There are patients who have cardiac failure from cor pulmonale as the symptom which first causes them to seek medical attention. By roentgenographic examination they may appear to have only a minimal amount of emphysema. However, on auscultation of the chest there are no breath sounds. Patients of this type are greatly benefited by surgery.

When the series was first studied it was thought that the disease would continue to progress and it would not be long before all the symptoms returned. This has not been the finding. Up to the present time the expected natural progression of the disease has not occurred after operation, even though the first patient in the group has been followed for six years. There appears to be no satisfactory explanation for this unexpected result.

Conclusions

Pulmonary emphysema is not an easily defined and concise disease entity. There are many variables.

Pulmonary emphysema is a condition that is found somewhere between bronchial asthma and localized cystic disease of the lung. When generalized pulmonary emphysema is present, all areas of the lung are not equally involved by tissue destruction. Some areas of the lung function better than others. Some areas have no function and interfere with the function of more nearly normal areas of the lung.

Surgery, by reducing the volume of the voluminous, overdistended, inelastic, emphysematous lung, causes it to fit the thoracic cage better. Thus, more nearly normal intrapleural negative pressures are restored and better motion of the chest cage and diaphragms are permitted. There is reduction in residual air. Denervation of the lung reduces bronchial secretions, reduces pulmonary artery pressure, and relaxes the bronchi. All of these factors improve ventilation and therefore relieve dyspnea.

Patients with pulmonary emphysema who do not obtain satisfactory relief of the symptoms by good medical treatment should be carefully considered for surgical treatment.

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This concludes the Saint Joseph's Hospital Issue of the MARYLAND STATE MEDICAL JOURNAL

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Special Articles

SOCIALIZED MEDICINE—APPROACHING AT A GALLOP—BY THE BACK DOOR

AMOS R. KOONTZ, M.D.

There are those in this country who believe that veterans of all wars and their dependents should be given free medical care in VA hospitals. Bills have been introduced into Congress which would effect this. So far as we know, none are pending in the present Congress. However, many bills have been introduced into the present Congress which would greatly broaden veterans benefits.

Furthermore, the bill for National Compulsory Health Insurance, which Harry Truman and Oscar Ewing tried so hard to get passed, has been re-introduced into the present Congress. The "do gooders," the political planners, and the believers in controlled economy are all behind it.

Little by little the believers in free enterprise, in the dignity of the individual, and in the principle of thrift and initiative, are losing ground and we are becoming more and more a paternalistic State. The Veterans Administration offers a wonderful opportunity for the believers in paternalism to make headway in a manner which is little noticed. According to the best available figures, 87 per cent of the patients in Veterans Hospitals now have non-service connected disabilities. I believe the figure to be actually higher. It can readily be seen that if free VA care is allowed to expand, and especially if it is allowed to include all veterans and their dependents, more than half of the population will be eligible for free medical care. What then would be the attitude of the rest of the population? It is obvious that under such circumstances it would be easy to pass the bill for National Compulsory Health Insurance.

The following article from the *Houston*, Texas, *Medical Record and Annals** for March 1955 gives a clear and concise statement of some of the things that are going on in VA hospitals. The VA is in competition, through care of workmen's compensation and insurance-covered cases, with physicians in private practice. Not only is the principle wrong, but it could be shown that it is highly expensive to the taxpayer. Every doctor should read the following highly instructive article from Texas:

"Numerous examples can be documented of Veterans' Administration hospitals admitting and treating non-indigent patients for conditions having no possible connection with service in the armed forces.

"The writer recalls several cases of hernia, incurred under coverage of the Workmen's Compensation Act of Texas, wherein the employee received a lump sum settlement including surgical fee, hospitalization expense and compensation for the convalescent period at the maximum weekly rate. When re-examined some six weeks later for return to duty, and found to have a good and strong repair, such cases readily volunteer the information that the surgery was done at the local VA hospital. And when asked how they were eligible for VA treatment since they claimed their disability to be due to recent industrial employment, and had been paid for by private industry, the answer is almost invariably a surprised-that-you-ask, 'I am a veteran.'

"The surgery is nearly always good, since it is often done by some of our well-trained colleagues who do private practice in honest competition, and on the side do part-time two afternoons or

* Reprinted by courtesy of *Houston*, Texas, *Medical Record and Annals*, March 1955.

days per week of staff work at the local VA hospital for fees of $25.00 to $50.00 per afternoon or day. It is possible these private practicing, part time VA colleagues don't do all of this surgery, but it is reasonably certain that VA authorities will not rush in to prove that much of it is being done by residents in training. It has been a long haul for the VA to sell the veterans on the fact that they are getting the best surgical, medical and dental care and the best social and other care.

"Now comes an incident of such concern to all practicing physicians. And the principle involved is of importance to every citizen of any occupation whatever.

"A VA hospital in Texas has presented a bill of $1,569.00 to a small industry's insurance carrier for services to an employee injured on duty in the small industry. The bill states it is for the first 54 days stay, and among the itemized parts are:

1. Anesthesia, $40.00.
2. Operation, removal of herniated nucleus pulposus bilaterally without laminectomy; spinal fusion, L5 to sacrum, $562.50.
3. Board and room to date 54 days at $14.75 per day, $796.50.

"In an accompanying letter and an interim summary, the VA hospital registrar shows full knowledge of the fact the veteran is not indigent, his condition is not service-connected, but is a responsibility of an insurance carrier under the Workmen's Compensation Act of Texas, and knowledge that the insurance carrier has already initiated treatment at the hands of private physicians of its choice.

"The patient had strained his back in the course of his employment in a small city and had gone to a chiropractor for treatment. Upon being given notice of injury, the carrier had the man removed to a larger city and placed under the care of a qualified orthopedic surgeon. The surgeon admitted the patient to a private hospital and began conservative treatment of traction in bed after routine x-rays, physical examination, etc. He also had consultation with a qualified neurosurgeon who noted the low back complaint, with pain in one buttock and thigh posterolaterally, but with no change from the normal in reflexes and no paresthesia. The neurosurgeon concurred in the continuing of conservative treatment.

"After 10 to 14 days in the hospital, the patient was allowed to go home, and was returned for treatment 2 weeks later. Readmission to the hospital was advised, but the patient refused. He wrote shortly thereafter that he was improving, but the next time he was heard from was after he was accepted in the VA hospital where, after 2 weeks period of conservative treatment, he was operated upon.

"The VA hospital advised it has placed a lien on 'any benefits that are due this veteran to pay the cost of his hospitalization here,' and had its 'chief attorney' file its claim with the Industrial Accident Board.

"From a study of this record, several conclusions must be drawn:

1. The so-called pauper's oath for veterans presenting themselves for treatment of non-service connected disabilities is knowingly disregarded by some VA hospitals.
2. The Federal Government, through its VA hospitals, is practicing Medicine and Surgery on individual private non-indigent patients, and is charging fees out of proportion (in their total) to what is ordinarily charged for similar services to a person covered by the Workmen's Compensation Act of Texas.
3. In listing the surgeon's and the anesthesiologist's fees, $562.50 and $40.00 respectively, the Federal Agency is either exploiting its full-time or part-time surgeons, or is paying them huge sums, while the tax-payer is paying for the operation of the hospital. Industry, through its insurance payments and taxes is, therefore, paying twice for one service.
4. In not communicating with the fully capable orthopedist and neurosurgeon who had begun treatment of the patient and in no way had relinquished his care, the VA showed poor professional practice, and the following borders on the unethical: A part of the VA history, published to the carrier and to be a part of the record before the Industrial Accident Board, contained the derogatory phrases, "His

treatment consisted of skin traction—and numerous pills of various sizes and shapes," and "became dissatisfied with his private physician and released himself from their care."

5. The Federal Government through its VA authorities is showing a total disregard for a ruling of the Industrial Accident Board of Texas in treating without authorization a case for which the carriers had already obligated itself to furnish treatment through capable surgeons of its choice and in a private hospital.

"Is it not time for county medical societies to take action along the only lines that can stop this well-advanced phase of federal practice of medicine? We, as local society members, aided whole-heartedly in the early phases of the present VA program when, just after the war, the great majority of patients had genuine service-connected disabilities, and we as veterans felt it our duty to help.

"We should now fight just as energetically a bureaucratic system that is actually using some of our private practicing members and our medical school deans in a way that will surely destroy the private practice of medicine if not stopped cold. AMA has already gone on record as opposing these abuses, but can do little on a national level. It has been belittled as a non-representative bunch of "brass hat" doctors in Chicago and Washington, out of touch with patients' problems. An honorable member of Congress recently told us the above was his evaluation of AMA.

"Some action along the following lines is imperative:

1. Request a complete and public census on all cases admitted to our local VA hospital during 1954, with a special reference to service-connected disability and ability to pay for private hospitalization; the cases to be listed by number so as to avoid embarrassment to many.

2. Offer to furnish the personnel for such a survey at the Society's expense, if expense of the survey is offered as an excuse.

3. Request our members who are primarily engaged in private practice to sever their connection with VA as soon as legally possible.

4. Request the Houston Dental Society to do likewise.

5. Request our members who are primarily engaged in private practice to discontinue any and all participation in the residency training program of the VA hospitals until all abuses are corrected.

6. Reassert ourselves in the operation of our own City-County Hospital for indigents, so that the ancillary services will be the best possible, whether the patients be ex-service men, ex-shipyard workers or whatever, so long as they need free care.

7. Urge our Industrial Accident Board to continue to disallow VA hospital bills, on the basis of its written letter to our Society recently wherein it stated unauthorized medical or surgical expense of a case under its jurisdiction would not be allowed.

8. Urge Insurance carriers, group hospitalization companies, etc., to rewrite their policies so as to exclude payment for VA hospitalization expense.

"It is later than we think. 'Creeping' is not the word for what is happening here. It is more fitting to use the description of our Congressman, who said he was glad to see a group of doctors looking so good (at a luncheon). He wanted to remember what we looked like before the freight train (Federal Medicine) ran over us.

"I think the Congressman's freight train can be stopped, but only with concerted local action; not by letting George (AMA) do it."

1014 St. Paul Street
Baltimore 2, Maryland

Component Medical Societies

BALTIMORE CITY MEDICAL SOCIETY

CONRAD ACTON, M.D.
Journal Correspondent

A special meeting of the Executive Board was called 4 April to consider 13 Recommendations recently transmitted by the Planning Committee of the Faculty. Dr. Robert Kimberly, our treasurer and representative on the Committee, lead the discussion of each point. After careful review, it was the unanimous opinion of the Board that the City Society itself should act upon them. A Special Meeting was decided upon and 26 April was selected as an appropriate date. Dr. John Classen, Secretary, was asked to arrange to have Faculty representatives present, if possible Drs. Warfield Firor, president; Wetherbee Fort, treasurer; and Everett Diggs, secretary. A meeting of the City Delegates was arranged, after the Special Meeting.

A communication was reported from the Ophthalmological Section of the City Society signed by Drs. F. M. Reese and H. K. Goldberg, representing the Committee for Proposed listing of Ophthalmologists in the Telephone Directory. The Section has determined to seek such listing. The executive Board feels that specialty listing should not be so used. It considers such listing to be an opening wedge for other specialty groups to seek the same thing, and disapproves it. Dr. Geraghty, president, pointed out that the Baltimore City Medical Society has no authority to select those who can be listed as specialists. The Secretary was directed to write Doctors Goldberg and Reese to this effect and to point out that the Executive Board considered it poor public relations. A copy of the letter was sent to the telephone company.

Intricacies of the Maryland Insurance Law rose to baffle us again. The letter from Dr. Geraghty, urging members to join the Group Malpractice plan, insured by Lloyd's of London, was interpreted by the State Insurance Commissioner as an attempt on the part of the Insurance Company to solicit business. There are two types of insurance carriers recognized by Maryland, apparently. Those with their funds invested in the State and those which have their funds invested without the State. Companies without capital funds in the State are not allowed to solicit business nor advertise in Maryland, according to the present law. What this will do to the Group Malpractice Plan is not yet clear. Possibly the circularization of the data with carrier's name "furnished on request" could avoid breaking the law.

Dr. Otto C. Phillips wrote concerning his amendments to the Constitution and By Laws. The secretary was asked to inform him that the amendments were in the hands of the proper Committee and that a special meeting was planned for April.

A Special Meeting of the Baltimore City Medical Society was held Friday, 26 April 1957 at 8:30 P.M. to consider the 13 Recommendations of the State Planning Committee formulated 20 March 1957. These Recommendations had been sent to all our Delegates, but were available to the members at large only when distributed at the Special Meeting. Attendance was very poor. Including at least five past presidents, twenty-seven members sat down when President Geraghty called the meeting to order, forty-two was the high tide, and thirty had stuck it out to adjournment. Dr. Everett Diggs, Faculty secretary, distilled the disappointment of the group, in the small number attending, when he said he had hoped there were more members "interested in a better return for the money they spend, but apparently not." Those present were in large part Delegates and Alternates anxious to serve the sentiment of the majority regarding the problems of the coming State meeting.

Dr. Albert E. Goldstein expressed regret that the "Recommendations" had not been sent in advance to all members with the announcement of the Special Meeting. "Then the members would know what the meeting was really about. They need to be told of the need for more money to run the State Organization right."

Dr. Amos Koontz rose to a point of order that there was no quorum present and we might as well go home now. Dr. Samuel Morrison (Delegate) urged that no matter how few were present they should have courage to take action. Dr. Harry

Klinefelter consulted the Constitution and By Laws up to and including the latest amendment and declared that a quorum was legally present.

Dr. A. S. Chalfant urged that the Recommendations be sent to all members to vote yes or no on an accompanying questionnaire. Dr. Ross Pierpont moved that since the membership was so uninformed, action should be delayed 1 year, and our Delegates instructed to endeavor to obtain deferment until careful consideration could be given. Another inquired, if the purpose was to aid in cutting expenses so that the annual assessments might be done away with, where in these Resolutions was there any economy? It was the consensus that there was none in fact. But it was also agreed that if an up and coming organization was wanted, it would have to be paid for.

Then each Recommendation was considered seriously; background provided by the State officers present was clarifying and most helpful. When the determining reasons for each action were known, the Meeting generally went along with the Recommendation. It disagreed with #7 regarding discontinuance of the Journal and emphatically voted to continue publishing it. Three Committees were voted to be saved from discard: Editorial Board of the Journal; Committee to Consider Relationship between Hospitals and Specialties and the Manner of Payment for Professional Services, and the Planning Committee, itself.

Regarding the proposed new medical building in Area 12, described in detail by Dr. Albert Goldstein, the Members voted that they felt no action on their part was called for. That the Delegates should act on it at a State level and consider it with regard to statewide suitability first.

FREDERICK COUNTY MEDICAL SOCIETY

LOUIS R. SCHOOLMAN

Journal Representative

The April meeting of the society was held at the Francis Scott Key Hotel. The speaker of the evening was Dr. Robert Crosby, pediatric neurosurgeon at the University Hospital, Baltimore. Dr. Crosby gave a most interesting informal talk on head injuries in children.

The medical colony gained two new members this month. A second child, David William, was born to Dr. and Mrs. Harry W. Gray. A fourth child, Lisa Ann, was born to Dr. and Mrs. A. M. Powell, Jr.

Hospital News

The April meeting of the Medical Staff was held on the first. After business had been transacted Dr. J. B. Thomas and Dr. T. E. Stone presented recent cases of lupus erythematosus.

The Clinico-pathologic Conference was held on April 9. The case was one of myasthenia gravis. Discussion was followed by a short review of the disease presented by Dr. Furie.

Health Departments

BALTIMORE CITY HEALTH DEPARTMENT

Summer Lead Poisoning in Children

The Commissioner of Health recently sent the following letter to all physicians in Baltimore:

"Dear Doctor:

Last year there were 48 cases of lead poisoning reported to us in young children in Baltimore City, and 3 of them died. Since 1930 there have been 548 of these cases, and 109 were fatal.

Lead poisoning can be prevented. A recent survey of dwelling units in the older and poorer sections of the City revealed that as high as 70 per cent of the paint samples taken from interior surfaces were positive for lead. The Health Department is making a concerted effort to have lead containing paints removed, but this will require time, maybe as much as ten years.

The incidence of lead poisoning reaches its peak in July and August; therefore, every physician is asked to be on the alert for suspected cases during the coming summer months. If illness does occur, it is important that the disease be recognized and treated early, otherwise it is likely to be fatal or cause permanent brain damage.

Unfortunately, there are no specific signs or symptoms, but if any child, especially in the 1–3 year old age group, shows any of the following symptoms, lead poisoning should be suspected:

1. Colicky abdominal pains
2. Persistent constipation
3. Anorexia
4. Anemia, resistant to iron therapy
5. Irritability or bizarre behavior pattern
6. Pica (Particularly paint and plaster)
7. Unexplained convulsion with or without fever
8. Frequent attacks of nausea and vomiting

The diagnosis can be established by blood lead determinations. The Bureau of Laboratories of the Baltimore City Health Department will do such tests and have available special lead free containers for collecting blood samples.

If, in your practice, you should find any cases of lead poisoning, I would appreciate your reporting the case to the Health Department on the Communicable Disease Report Card. Home investigation can then be carried out promptly to determine the source of the lead poisoning, and thereby the condition can be corrected to prevent recurrences.

Your help and interest will be deeply appreciated.

Cordially yours,

Huntington Williams, M.D.

Commissioner of Health"

STATE DEPARTMENT OF HEALTH

Today's Role of Epidemiology in Medicine

The Division of Epidemiology and Communicable Disease Control, a reorganized unit of the Maryland State Department of Health, was recently established and Dr. Charlotte Silverman was appointed its first chief on October 17, 1956. The work which this division will pursue relates to the epidemiologic aspects of various types of disease and health problems. While epidemiology was originally concerned with epidemics and has been traditionally associated with communicable diseases, it has become evident that the epidemiologic approach is suitable and useful for the fuller understanding of other conditions such as chronic non-infectious diseases, accidental injuries, and mental illness.

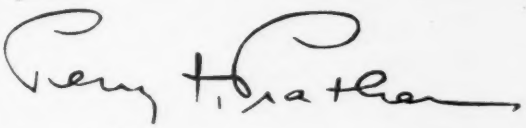
By techniques such as field and record studies and microbiologic investigations, the epidemiologist tries to determine factors related to susceptibility to disease and means of transmission. In this way he hopes to bring to light etiologic agents, and thereby ultimately to control disease. In contrast to the practitioner of clinical medicine who is concerned primarily with individual cases, the epidemiologist deals with cases as a group in the population. Illness or death are related to the group in which these events occur. This approach can reveal information or suggest methods of inquiry

which are generally not possible through the study of individual cases.

The epidemiologic approach may often lead to findings which are quite different from those derived from hospital experience or from the experience of practicing physicians. This is to be expected because the epidemiologist seeks to identify *all* cases of a disease, not only the clinical cases but the subclinical, atypical, and undiagnosed as well. Knowledge of a disease such as tuberculosis, its course and outlook, is incomplete if the symptomless, healed and unknown cases are not a part of the study population.

A constant feature of epidemiologic work is the search for population groups with high rates of disease and with low rates, so that the reasons for disease or absence of disease can be discovered. Morbidity studies always involve the determination of age and sex incidence of disease, a classical contribution of this method. Mortality trends can be more clearly interpreted by means of the epidemiologic approach when an apparent increase in deaths may reflect either a true increase, as appears to be the case in leukemia, or the effects of better recognition and diagnosis of the disease combined with improved diagnostic facilities, as seems likely with lead poisoning in children. The epidemiologic method has been helpful in separating entities that were formerly grouped together, such as the separation of peptic ulcers into gastric and duodenal ulcers, and it is hoped it may contribute to the clear distinction of coronary arteriosclerosis from other types of coronary heart disease. The reverse process has shown that apparently different conditions were related, as in the case of streptococcal infections which now include rheumatic fever as well as scarlet fever, streptococcal sore throat, erysipelas and puerperal infection.

Accurate diagnosis is basic to sound epidemiologic work because of the need to identify inapparent cases as well as those of clinical interest, and because of the importance of separating population groups with a given disease from those which are free of the disease. Our efforts to combat disease and promote health can be most productive if clinician, laboratory investigator and epidemiologist work closely together.



Director

NEW TYPE-A INFLUENZA

AMA News Release

Doctors are asked by the Public Health Service to be on the alert for a new type-A influenza strain expected to work its way into this country from the Far East. Details may be obtained from state health departments.

STATE OF MARYLAND DEPARTMENT OF HEALTH

MONTHLY COMMUNICABLE DISEASE REPORT

Case Reports Received during 4-week Period, June 1–27, 1957

	CHICKENPOX	DIPHTHERIA	GERMAN MEASLES	HEPATITIS, INFECT. AND SERUM	MEASLES	MENINGITIS, MENINGOCOCCUS	MUMPS	POLIOMYELITIS, PARALYTIC	POLIOMYELITIS, NON-PARALYTIC	ROCKY MT. SPOTTED FEVER	STREP. SORE THROAT INCL. SCARLET FEVER	TYPHOID FEVER	WHOOPING COUGH	TUBERCULOSIS, RESPIRATORY	SYPHILIS, PRIMARY AND SECONDARY	GONORRHEA	OTHER DISEASES	DEATHS: Influenza and pneumonia
Total, 4 weeks																		
Local areas																		
Baltimore County	13	—	—	—	9	—	33	—	—	—	7	—	12	13	—	2	m-1	3
Anne Arundel	—	—	1	—	9	1	6	—	—	1	2	—	—	4	—	4	—	3
Howard	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Harford	1	—	1	—	1	—	2	—	—	1	—	—	—	4	—	—	—	—
Carroll	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—
Frederick	2	—	4	1	4	—	23	—	—	—	1	—	2	1	—	1	e-1	4
Washington	—	—	—	1	—	—	—	—	—	—	—	—	—	3	—	—	—	—
Allegany	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	6
Garrett	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Montgomery	16	—	7	1	57	1	46	—	—	—	42	—	2	3	—	—	—	5
Prince George's	1	—	8	1	7	—	8	2	—	1	3	—	—	6	—	2	m-2	5
Calvert	2	—	—	—	1	—	—	—	—	—	—	—	1	—	—	—	—	1
Charles	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—
Saint Mary's	—	—	2	—	1	—	3	—	—	—	—	—	—	2	2	—	—	1
Cecil	1	—	—	—	—	—	—	—	—	—	1	—	—	2	—	—	—	1
Kent	7	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—
Queen Anne's	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—
Caroline	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Talbot	—	—	—	—	—	—	2	—	—	—	—	—	—	4	—	3	—	2
Dorchester	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1
Wicomico	3	—	1	3	8	—	2	—	—	—	3	—	—	1	—	6	—	1
Worcester	—	—	—	—	12	—	1	—	—	—	—	—	—	1	—	—	—	1
Somerset	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—	—
Total Counties	46	0	24	7	110	2	127	3	0	3	59	0	17	51	3*	18		34
Baltimore City	72	0	13	1	83	1	100	1	0	0	12	0	18	87	12	558		21
State																		
June 1–27, 1957	118	0	37	8	193	3	227	4	0	3	71	0	35	138	15	576		55
Same period 1956	217	0	189	9	460	6	328	6	3	4	39	2	7	165	19	615		34
5-year median	238	1	55	25	420	2	202	5		4	67	2	36	169	16	665		29
Cumulative totals																		
State																		
Year 1957 to date	1803	0	212	75	820	18	1770	4	0	6	715	1	154	873	109	3346		377
Same period 1956	2316	0	977	66	9030	36	2243	7	3	6	605	8	80	1090	152	3268		413
5-year median	2923	7	472	199	5580	29	1564	9		6	1194	9	183	1094	106	3578		400

e = encephalitis from mumps
m = meningitis, other than meningococcus
* Total includes cases reported by State Hospitals and Institutions